

## JK-PRACTITIONER

Volume 14 Number 2,  
April-June 2007  
ISSN No. 0971-8834

### EDITOR-IN-CHIEF

GM Malik MD,FACG  
PO BOX 884,Srinagar-190001  
(INDIA), Mobile 9419007198

### CONSULTANT EDITORS

USA:

Farooque A Khan MB,MACP,FCCP; Arfa Khan MD;Muzaffar Ahmad MD;PK Shah MD,FACS;Fiyaz A Shawl MD;Farooq Peerzada MD;Ramesh Khordari MD,FACP,FACE;M. Yusuf Khan FCAP; Ghulam Jeelani Dhar MD,FACG;Khalid J Qazi MD,FACP;Ifthikar J Shah MD,FACC;MA Shah MD,FACG;Gh.Nabi Mir B.Sc.M Pharm,Ph.D;Abdul Rouf Mir MD UK

Altaf Hussain MS,MCh Orth;FICS GJ Mufti DM,FRCP;GR Mufti FRCS;MA Mir FRCS  
KSA

Mohammad Al Karawal FA;Ibrahim Al Tememi FA;Abdul Majid FRCS;Fadel A Razaak FRCS;Syed Manzoor Qazi FRCS;Saleh M Al Salamah FRCS;Shabir A Qazi MS  
INDIA

S Nundy MS,MCh; Naresh Tehran; MCh, Upendra Koul MD,DM,DNB; BN Tandon, Rakesh Tandon MD, PhD; VP Venktachanlam MS; (New Delhi)Ketan Desai MS,MCh;(UP) MS;S Sodan MS(Jammu)Sudipo Roy MD (West Bengal);S Malhotra MD,DM (Chandigarh);MS KhurooMD,DM; S. Jalal DM; M. Afzal Wani, Ashiq H. Naqshbandi (Srinagar)

### ASSOCIATE EDITORS:

Mir Tanveer (USA); MA Jalu (UK) RK Chrungeo (Jammu) MA Kamili;Muzaffar Ahmad(Srinagar)

### DEPUTY EDITORS:

Riaz Bashir (Boston); Showkat Bashir (NY)Malik Suhail (Srinagar)

### ASSISTANT EDITORS:

Gulzar A Hagroo (UK);Abdul Rashid Rather,Iqbal Saleem Mir (JK)

### ADVERTISING EXECUTIVES:

ML Bhat,B Ahmed(Srinagar)  
Irfan (Jammu)

### MANAGING EDITOR:

Malik Suhail

### ADVISORY BOARD

SN Ahmad Shah FRCP; S Zahoor Ahmad FRCP GQ Allaqaband FRCP; AR Khan MD; Sheetal Singh; Girja Dhar MS; Mahmooda Khan MS; MA Wani Mch; N.A. Wani MS; G.R. Mir MS; T.S. Sethi MS; A Parihar DM; S. Abdul Ahad MS; Abdul Kabir Dar MD (ISM) MS; Abdul Aziz Hagroo(KSA); Rafiq Shiekh,Shagufta Yasmeen,(USA);Vijay Gupta, Jashbir Singh(Jammu);Khurshed Iqbal,Mushtaq A Magroob,Riaz Farooq,Qazi Mushtaq Ahmad(Srinagar)

Designing & Setting : Naushad Ali

### Commentary

- 110 Lateral Decubitus Position in Spinal Surgery - Current Concepts  
*Suhail Afzal, Asif Sultan, Mohd Iqbal & Shabir Ahmad Dhar*

### Clinical Image

- 113 Doxycycline Induced Photo Onycholysis and Nail Pigmentation  
*Qazi Masood Ahmad, Iffat Hassan Shah, Qazi Nuaman Masood & Farah Sameem*

### Epidemiology

- 114 The Epidemiology of Fractures and Dislocations at District Hospital Kathua (J&K)  
*Rakesh Sharma, Liaqat Ali & Mukul Ubbot*

### Essay

- 118 A Sociological peresctive on Kashmiri women in medical profession  
*Shamshad & B. A. Dabla*

### Students Page

- 122 Vital statistics in pulmonary embolism  
*Tabassum Parvez*

## JK-PRACTITIONER

## Volume 14, Number 2

### AIMS AND SCOPE:-

JK-Practitioner is a multidisciplinary Medical Journal published quarterly from Srinagar.Emphasis is placed on matters related to medicine in the country in particular,while articles from anywhere in the world are given due importance.Besides original articles and case reports the journal includes clinical reviews,guest articles and special articles on current topics in medicine and therapeutics.

### INFORMATION FOR SUBSCRIBERS:-

The JK-PRACTITIONER-Frequency Quarterly,one volume per year beginning in January.Other issues in April,July and October.**Correspondence regarding Editorial matters** should be addressed to the Chief Editor,GM Malik,PO BOX No 884,GPO,Srinagar, Kashmir (INDIA).**Annual Subscription rates:-Personal Rs.500/-Institutional Rs.700/-, Reduced Rs.400/- Institutional rate** applies to Libraries, Schools, Hospitals, Clinics, Commercial and Private institutions and organisations.**Reduced rate** is available for medical students only.**Rates outside India** US \$100/-Gulf countries SR 100/-All other countries US \$100 or equivalent.**Concessional Rate for personal subscription** is available for a three year term at Rs.1400/- Outside India US \$ 200/- Gulf Countries SR 250/-Postal Charges within the country are Rs.50/-extra for a one year subscription.**Single current issue** Rs.125/- **Prices are subject to change.****Change of Address:-** The publisher must be notified 60 days in advance.**Volume Index** appears in October issue.**Indexing/Abstracting Services:-**The journal is currently abstracted and indexed in EMBASE/Excerpta Medica database,IndMED, Pubmed (Medline) and listed in Ulrich's Periodicals Directory (USA), INSDOC (INDIA). And Index Academicus (IA). **Copyright information:-**When the manuscript is accepted for publication the authors agree to automatic transfer of the copyright to the publisher that the manuscript will not be published elsewhere in any language without the consent of the copyright holder.Old issues of the journal can be purchased at 15 % discount subject to availability.

### INFORMATION FOR AUTHORS SUBMITTING MANUSCRIPTS:-

Manuscripts are to be submitted on Compact Disk in MS Word with two hard copies\*.The articles undergo the standard review process.Please allow 4-6 weeks before information regarding acceptability,resubmission or rejection is conveyed to the authors.Once the article is cleared for publication the authors will have to bear the publication cost as Under:-  
Reviews\Original articles\Case reports Rs.1200/-Commentary,Research Letter etc Rs.1000/-  
Clinical Image Rs.800/- Other articles Rs.700/- Photographs come at an extra cost of Rs.200/- per B&W print and Rs.500/- per colour print.More than three tables Rs.100/table

**WEBSITE** <http://www.indmed.nic.in>,

\*To speed up the process of REVIEW, manuscripts could be submitted on [zabbask2003@yahoo.co.in](mailto:zabbask2003@yahoo.co.in) with intimation to [zaffarak@hotmail.com](mailto:zaffarak@hotmail.com)

JK-Practitioner Volume 14 Number 2, April- June 2007 Year 14, Issue 51.



done *Low myopia*, characterized by a myopic refractive error less than -3 *Medium myopia*, characterized by a myopic refractive error of -3D to <-6D *High myopia*, with refractive power more than -6D.

#### Ocular manifestations of degenerative myopia.

(a) **Anatomical manifestations:** The more important anatomical abnormalities found in extremely myopic individuals include increased axial length, corneal astigmatism, deep anterior chamber, angle iris processes, zonular dehiscences, vitreous syneresis, lattice retinal degeneration, scleral expansion and thinning, decreased ocular rigidity, posterior staphyloma, tilted disc, haloatropy, macular lacquer cracks, pigment epithelial thinning, choroidal attenuation, myopic crescent, anular crescent, post staphyloma, Lacquer cracks, Elscling pearls, lattice degeneration, retinal holes, retinal tears, Hoishe shoe tears, vitro retinal tractions, snow flake degeneration, paring stone degeneration etc.

(b) **Functional manifestations:** These include image minification, anisometropic amblyopia, subnormal visual acuity, visual field defects, impaired dark adaptation, abnormal color discrimination and suboptimal binocularity.

#### Treatment Options

##### a. Optical Correction

Optical correction in the form of spectacles or contact lenses provides clear distance vision. Eyeglasses are the most common method used to correct myopia. Concave glass or plastic lenses are placed in frames in front of the eyes. The lenses are ground to the thickness and curvature specified in the eyeglass prescription. The lenses cause the light rays to diverge so that they focus further back, directly on the retina, producing clear distance vision.

Whether spectacles or contact lenses are preferable in a given case depends upon numerous factors, including patient age, motivation for wearing contact lenses, compliance with contact lens care procedures, corneal physiology, and financial considerations.

##### b. Refractive Surgery

Refractive surgery is evolving field of ophthalmology focusing on the correction of refractive errors of eye including myopia. The ultimate goal of refractive surgery is to obtain vision that is quantitatively and qualitatively better than that which can be obtained using spectacles and contact lenses with a procedure that is adjustable and reversible. There are several refractive surgery methods in use; others are in various stages of research and development.

##### Radial Keratotomy (RK):

In radial keratotomy (RK), a spoke-like radial pattern of incisions in the paracentral cornea weaken a portion of the cornea. The weakened part steepens while the central cornea flattens. The amount of resultant refractive change depends on the size of the optical zone and the number and depth of the incisions. The method is used to treat myopia in the range of -2.0 to -6.0 D. The reported visual complications of RK include diurnal variation of refraction and visual acuity, glare, monocular diplopia, a presumably permanent reduction in best corrected visual acuity, increased astigmatism, irregular astigmatism, induced anisometropia, and a gradual shift toward hyperopia that continues for months or years after the surgical

procedure.<sup>11</sup>

This shift toward hyperopia can lead to early onset of presbyopic symptoms. RK incisions may also decrease the structural integrity of the globe.<sup>12</sup>

(ii) *Excimer laser photorefractive keratectomy (PRK):* The most recent development in vision correction is a procedure called Photorefractive Keratectomy or PRK. Although the approach is similar to RK, in that the cornea is modified to correct vision, the process is vastly different with remarkable improvements in patient risk and correction capabilities. Rather than making cuts in the cornea, the PRK process uses an excimer laser to sculpt an area 5 to 9 millimeters in diameter on the surface of the eye. This process removes only 5-10% of the thickness of the cornea for mild to moderate myopia and up to 30% for extreme myopia. The major benefit of this procedure is that the integrity and the strength of the corneal dome is retained. The excimer laser is set at a wavelength of 193nm, which can remove a microscopic corneal cell layer without damaging any adjoining cells. This allows the practitioner to make extremely accurate and specific modifications to the cornea with little trauma to the eye. This ability to sculpt, rather than cut, opens up the arena for treating additional vision conditions. At this stage, there are excimer laser machines that with a combination of masks and computer controls, can reliably treat myopia, hyperopia and now astigmatism. The method is used to treat myopia in the range of -1.0 to -6.0 D. Best results are achieved for myopia of up to -3.5 D. Possible problems include delayed surface healing, corneal haze and or scarring, over or undercorrection, and the development of astigmatism. Some individuals can have a poor or excessive healing response.

*Laser In Situ Keratomileusis (LASIK):* It is a procedure in which an anterior flap of cornea is lifted with a keratome and an excimer laser is used to sculpt the stromal bed to change the refractive error of eye. It can be used to correct 0.5-12D of myopia. Despite the overwhelming success of LASIK, complications of LASIK may result in poor visual outcomes. LASIK complications include free cap, incomplete flap, buttonhole, thin flap, rregular flap, flap dislocation, and intraocular penetration.

*Laser Subepithelial Keratomileusis (LASEK):* It is simple, inexpensive procedure that involves the creation of an epithelial flap after exposure to trephine, 18% alcohol, for 25 seconds and subsequent replacement of flap after laser ablation.<sup>13,14</sup> The method is used to treat myopia in the range of -1.0 to -12.0 D (or even up to -15 D)<sup>15</sup>

##### Prevalence

Myopia is the most common eye disorder and is significant health problem associated with increased risk of visual loss around the world<sup>16</sup>. Myopic chorioretinal degeneration is the fourth most frequent cause of blindness and accounts for 8.8% of all causes<sup>17</sup>. The prevalence of myopia varies by country and by ethnic group, reaching as high as 70-90% in some Asian populations<sup>18</sup>. In Europe and America its prevalence varies between 30-40% while in Africa 10-20% of the population is affected<sup>19</sup>. Myopia affects 25% population in United States<sup>20</sup>. It may exceed 65% in Chinese or Indians<sup>21</sup>. Our unpublished data indicates 4.87% prevalence in school going students in Srinagar city (IA, TQ and KIA-Unpublished data)

High myopia affects 27-33% of all myopic eyes

corresponding to prevalence of 1.7-2% in general population in United States<sup>21</sup>. In Japan high myopia reportedly affects 6-18% of myopes and 1-2% of general population<sup>22</sup>. Prevalence of high myopia in France is 3.2% and up to 9.6% in Spain<sup>23</sup>.

#### **Ethnic diversity affects myopia**

Ethnic diversity appears to distinguish different groups with regard to prevalence. Caucasians seem to have a higher prevalence than African Americans<sup>24</sup>. Jewish Caucasians have been found to have higher myopia prevalence than general Caucasian population in both US and European population surveys; orthodox Jewish males in particular show increased susceptibility<sup>25</sup>.

#### **Etiology**

One of the oldest arguments surrounding myopia is whether the condition results from hereditary factors or environmental influences such as reading, close work, accommodation and diet. There is strong evidence that both heredity and environment play a role in the development of myopia and the "nature vs. nurture" debate has been on for a while.<sup>26</sup>

**Environmental influences:** There are various evidences, which prove that environment plays a role in the development of myopia. Environmental factors, such as educational level, occupation, and individual income, have been associated with the prevalence of myopia<sup>27</sup>. Other personal factors, such as reading habits and use of computers, may also affect the progression to high myopia<sup>28</sup>. Occupational groups with prolonged hours of close up work such as microscopists and carpet weavers have a higher incidence of adult-onset myopia and higher rates of myopia progression in adulthood<sup>29,30</sup>. The correlation between level of academic achievement and the prevalence and progress of myopic refractive errors is strong; people whose professions entail much reading during either training or performance of the occupation (lawyers, physicians, microscopists, and editors) have higher degrees of myopia and the myopia may progress not just in people's teenage years but throughout their 20's and 30's<sup>31</sup>.

The rapid increase in the prevalence of myopia over the last several decades suggests that environmental factors are important<sup>32</sup>. Cordain et al<sup>33</sup> suggest a positive correlation for myopia with increased consumption of carbohydrates, hyperinsulinemia and type II diabetes.

**Animal models of myopia:** Modern research on animals leaves no room for doubt that ultimate shape of the eye can be severely influenced by environmental factors. Whether using primates (monkeys, marmosets or tree shrews) or chickens, investigators have shown that when a clear formed image is not allowed to be focused on retina (by saturating up eye lids or placement of translucent goggles or minus diopter contact lenses) high myopia will develop in the eyes of young animals<sup>34</sup>. Before we conclude that myopia in humans is analogous to experimentally induced myopia in animal models, we should bear in mind that naturally occurring disease processes causing deprivation of formed vision do affect human infants. In periorbital haemangiomas and congenital cataracts, the two conditions that have been most studied, occlusion of visual axis occurs in first few months of life<sup>35</sup>. In eyes that are not treated promptly, axial elongation and myopia develop. Other conditions that are

associated with myopia include congenital ptosis, perinatal, vitreal and retinal haemorrhages and inflammatory keratitis. These naturally occurring experiments of deprivation of formed vision are consistent with animal models previously described.

**Genetic predisposition:** Despite the many years of research, little is known about the precise molecular defects and abnormal biochemical pathways that result in myopia. Compelling data from familial aggregation and twin studies indicate that susceptibility to myopia is inherited. Several familial aggregation studies have reported greater prevalence of myopia in children of myopic parents compared to children of non-myopic parents<sup>36,37</sup>. Several twin studies have demonstrated a very high heritability for myopia. Other recent genetic studies of families with high myopia have reported significant linkage to regions on chromosome X (MYP1), 18p11.31 (MYP2), 12q23.1-24 (MYP3), 17q21-22, 7q36 and 2q37.1<sup>38-41</sup>. The 18p candidate region has been confirmed in an independent study of high myopia<sup>42</sup>. Mutti et al<sup>43</sup> examined the hypothesis that families with milder, juvenile onset myopia may show linkage to these same candidate regions. They found no evidence to support such a role in this more common form of myopia but their study was not so powered in presence of heterogeneity.

#### **Candidate genes**

The direct analysis of the sequence within a critical region can be the most accurate, precise and efficient approach to the disease gene identification. This is particularly true for instances where the 'perfect' candidate gene (based on function or expression) does not exist within a defined critical region. It is true for a disorder such as myopia, in which temporal and spatial expression of the disease gene is not known, and could be restricted to early development and to any eye component. All genes that map within the 18p11.3 (MYP2) critical region are therefore candidate disease genes based on position.

There are 9 known genes that are considered candidates based on mapped position within the MYP2 interval. These 9 genes include clustrin-like 1 (CLU1), elastin microfibril interfacer 2 (EMILIN 2), lipin 2 (LPIN 2), myomesin 1 (MYOM1), myosin regulatory light chain 3 (MRCL3), myosin regulatory light chain 2 (MRLC 2), transforming growth factor  $\alpha$ -induced factor (TGIF  $\alpha$ ), large Drosophila homolog associated protein 1 (DLGAP1) and zinc finger protein 161 homolog (ZFP 161). Mutation analysis of these 9 encoded positional candidate genes did not identify sequence alterations associated with the disease phenotype<sup>44</sup>. Similarly Lumican (LUM) and Fibromodulin (FMOD) have been excluded as candidate genes in MYP3 associated high grade myopia<sup>45</sup>.

TGIF was earlier demonstrated to be candidate gene for high myopia<sup>46</sup>. TGIF is a DNA binding homeodomain protein that belongs to TALE homeobox family<sup>47,48</sup>. It is a transcription repressor with multiple actions, including a role in retinoid responsive transcription<sup>49</sup>. TGIF mutations are associated with holoprosencephaly, a congenital craniofacial and brain anomaly disorder<sup>50-52,53,54</sup>.

#### **Discussion**

The observations establishing the linkage of several loci with high myopia may be indicative of the involvement of more than one gene in the disease process. It is likely, that such an association may simply reflect variants of the disease rather than any ethnic diversity at genetic level. Furthermore, the parameters defining high myopia being less stringent have contributed to the diverse picture. However, genetic complexities associated with several disorders strengthen the possibility of high myopia being a multigenic disorder. Several chromosomal regions have been attributed to harbor the potential candidate gene for high myopia, a few of them implicated without any authentic attribution to the genetics of the disease. The only substantial evidence for the candidature thus far, may have been obtained for TGIF gene with several SNPs reported<sup>21</sup>. Such polymorphisms have more often than none reflected the variations in one or a set of ethnic populations than being of general significance with regard to disease attribution. Such data nonetheless has oriented the focus of disease genetics more toward the origin of the population rather than the size.

We are attempting to investigate a cohort of two and three generation high myopic families from across Kashmir region for genetic studies. In an ideal scenario a larger family

with nine or more individuals affected would be more informative. Despite this limitation, the ethnic background associated with even smaller families would be of immense significance in terms of the genetic evaluation and comparison to other studies taken up globally. We are also trying to substantiate the relevance of loci believed to be associated with the disease and verifying the authenticity of genes like TGIF that have been implicated, though controversially, in the disease process.

### Conclusion

Myopia is a huge public health problem worldwide. Severe myopia is a cause of visual disability and leads to vision impairment and blindness. In addition to human cost of visual disability there is profound economic cost on society. Substantial resources are required for optical correction of myopia with spectacles, contact lenses and surgical procedures. Since no current treatments can reverse the structural changes of pathological myopia, preventing myopia is a challenge before scientists researching vision. Understanding the mechanisms and factors that affect ocular growth is prerequisite to the development of therapeutic strategies.

## References

- Grosvenor T. "A review and a suggested classification system for myopia on the basis of age-related prevalence and age of onset." *Am J Optom Physiol Opt*. 1987 Jul;64(7):545-54
- Goss DA, Eskridge JB. Myopia. In: Amos JF, ed. *Diagnosis and management in vision care*. Boston: Butterworths, 1987:121-71.
- Curtin BJ. The myopias: basic science and clinical management. Philadelphia: Harper & Row, 1985:237-435
- Locke LC. Induced refractive and visual changes. In: Amos JF, Yanhoff M, and Duker J.S. in *Ophthalmology*, second edition, 2006, vol 2, p. 934-935
- Waring GO III, Lynn MJ, Golender H, et al. Results of the prospective evaluation of radial keratotomy (PERK) study one year after surgery. *Ophthalmology* 1985; 92:177-99.
- Grosvenor T. How predictable are the results of excimer laser photorefractive keratectomy? A review. *Optom Vis Sci* 1995; 72:698-712
- Hoffer KJ, Darrin JJ, Pettit TH, et al. Three years experience with radial keratotomy—the UCLA Study. *Ophthalmology* 1983; 90:627-36.
- Bores LD. Historical review and clinical results of radial keratotomy. *Int Ophthalmol Clin* 1983; 23:93-118.
- Schachar RA. Indications, techniques, and complications of radial keratotomy. *Int Ophthalmol Clin* 1983; 23:119-28.
- Binder PS. Optical problems following refractive surgery. *Ophthalmology* 1986; 93:739-45.
- Waring GO III, Lynn MJ, Culbertson W, et al. Three-year results of the prospective evaluation of radial keratotomy (PERK) study. *Ophthalmology* 1987; 94:1339-53.
- Duling K, Wick B. Binocular vision complications after radial keratotomy. *Am J Optom Physiol Opt* 1988; 65:215-23.
- Bullimore MA, Sheedy JE, Owen D. diurnal visual changes in radial keratotomy: implications for visual standards. *Optom Vis Sci* 1994; 71:516-21.
- Waring GO III, Lynn MJ, McDonnell PJ. Results of the prospective evaluation of radial keratotomy (PERK) study 10 years after surgery. *Arch Ophthalmol* 1994; 112:1298-308.
- Vinger PF, Mieler WF, Oestreicher JH, et al. Ruptured globes following radial and hexagonal keratotomy surgery. *Arch Ophthalmol* 1996; 114:129-34.
- Azar DT, Ang RT, Lee JB, et al., Laser subepithelial kerato: leusis: electron microscopy and visual outcomes of flap PRK. *Curr Opin Ophthalmol*, 2001, 12, 323-328
- Azar DT, Ang RT, Laser subepithelial keratomileusis: evolution of alcohol assisted flap surface ablation. *Int Ophthalmol Clin*, 2002, 42, 89-97
- Feit R, Taneri S, Chen CC, et al., LASEK techniques and outcomes. *Ophthalmol Clin North Am*. In Press
- Ditzen K, Huschka H, Laser in situ keratomileusis (LASIK) for myopia, in *Proceedings of the 6th International Conference on Myopia*, Springer, Tokyo 1998, p. 169
- Fredrick DR. *Myopia*. *BMJ* 2002, 324, 1195-99
- National Advisory Eye Council; Strabismus, Amblyopia and visual processing Panel, Vision Research: A national plan, 1999-2003, Washington D.C; National Institute of Health, NIH Publication, 98-4120
- Curtin BJ; Basic Science and Clinical management. In *The Myopias*, New York, Harper and Row, 1985, 237-245
- Leibowitz HM, Krueger DF, Maumder LR, The Framingham Eye study monograph, *Surv Ophthalmol*, 24 (1980) 472-479.
- Katz J, Tielsch JM, Sommer A, Prevalence and risk factors for refractive errors in an adult inner city

- population, *Invest Ophthalmol Vis Sci*, 38(1997)334-340.
26. Burton TC, The influence of refractive errors and lattice degeneration on the incidence of RD, *Trans Am Ophthalmol Soc*, 87(1990)143-155
  27. Curtin BJ, Myopia: A review of its etiology, pathogenesis and treatment, *Surv Ophthalmol*, 15(1970)1-17.
  28. Ghafour IM, Allan D, Foulds WS, Common causes of blindness and visual handicap in the west of Scotland, *Br J Ophthalmol*, 67(1983)209-213
  29. National Advisory Eye Council (US), Vision Research: a National Plan, 1983-1987, Washington, DC, US Department of Health and Human Service, National Institutes of Health (1982).
  30. Fuchs A, Frequency of myopia gravis, *Am J Ophthalmol*, 49 (1960)1418.
  31. Baldwin W; A review of statistical studies of relations between myopia and ethnic, behavioral and physiological characteristics, *Am J Opt Physiol Optics*, 58 (1981) 516-527
  32. Zylbermann R, Landau D, Berson D; The influence of study habits on myopia in jewish teenagers, *J Pediatr Ophthalmol Strab*, 30 (1993)319-322
  33. Mutti DO, Zadnik K, Adams AJ, Myopia: The nature vs nurture debate goes on, *Invest ophthalmol Vis Sci*, 37(1996)952-957
  34. Mutti DO, Zadnik K, Is computer use a risk factor for myopia, *J Am Optom Assoc*, 67(1996)521-30
  35. Gobba FM, Broglia A, Sarti R, Luberto F, Cavalleri A, Visual fatigue in video display terminal operators: Objective measure and relation to environmental conditions, *Int Arch Occup Environ Health*, 60(1988)81-87
  36. Adams DW, McBrien NA, Prevalence of myopia and myopic progression in a population of clinical microscopists, *Optom Vis Sci*, 69(1992)467-473
  37. Tay MT, AU Eong KG, Ng GY, Lim MK, Myopia and educational attainment in 421,116 young Singaporean males, *Ann Acad Ed Singapore*, 21(1992)785-789
  38. Saw S, Neito FJ, Katz J, Schein OD, Levy B, Chew S, Factors related to the progression of myopia in Singaporean children, 77 (2000) 549-554
  39. Teasdale TW, Schmidt E, Myopia and its relationship to education, intelligence and height preliminary results from an ongoing study of Danish dralies, *Acta Ophthalmol Suppl*, 185(1988)41-43
  40. Dunphy EB, Stoll MR, King SH, Myopia among American male graduate students, *Am J Ophthalmol*, 65(1968)518-521
  41. Cordain L, Eaton SB, Brand Miller J, Linderberg S, Jensen C: An evolutionary analysis of the aetiology and pathogenesis of juvenile onset of myopia, *Acta Ophthalmol Scand*, 80 (2002) 125-135
  42. Hammond CJ, Snieder H, Gilbert CE, Spector TD: Genes and environment in refractive error: the twin eye study, *Invest Ophthalmol Vis Sci*, 42(2001)1232-1236
  43. Cordain L, Eaton SB, Brand Miller J, Linderberg S, Jensen C: An evolutionary analysis of the aetiology and pathogenesis of juvenile onset of myopia, *Acta Ophthalmol Scand*, 2 (2002) 125-135
  44. Raviola F, Wiesel TN, An animal model of myopia, *N Engl J Med*, 312(1995,)1609-1615
  45. Wallmain J, Turkel J, Trachmann J; Extreme myopia produced by modest change in early visual experience, *Science*, 201 (1978) 1240-51
  46. Siegwart JT Jr, Norton TT, The susceptible period for deprivation induced myopia in tree shrew, *Vision Res*, 38(1998,)3505-3515
  47. Whatham AR, Judge S, Compensatory changes in eye growth and refraction induced by daily wear of soft contact lenses in young marmosets, *Vision Res*, 41, 2002)267
  48. Hoyt CS, Stone RD, Fromer C, Billson FA, Monocular axial myopia associated with neonatal eyelid closure in human infants, *Am J Ophthalmol*, 91(1981)197-200
  49. Robb RM, Refractive errors associated with hemangiomas of the eyelids and orbit in infancy, *Am J Ophthalmol*, 83 (1977,) 52-58
  50. Krause UH, Rantakallio PT, Koironen MJ, Mottonen JK, The development of myopia upto the age of twenty and a comparison of refraction in parents and children, *Arctic Med Res*, 52(1993)161-165
  51. Hui J, Peck L & Howland HC, Correlation between familial refractive error and children's noncycloplegic refractions, *Vis Res*, 35(1995,)1353-1358
  52. Sorsby A, Leary GA & Fraser GR, Family studies on ocular refraction and its components, *J Med Genet*, 3 (1966)269-273
  53. Keller JT; A comparison of the refraction of myopic children and their parents, *Am J Optom Arch Am Acad Optom*, 50(1973)206-211
  54. Yap M, Wu M, Liu ZM, Lee FL & Wang SH, Role of heredity in the genesis of myopia, *Ophthalmol Physical Opt*, 13(1993)316-319
  55. Schwartz M, Haim M & Skarsholm D, X-linked myopia: Bornholm eye disease- linkage to DNA markers on the distal part of Xq, *Clin Genet*, 38 (1990)281-286
  56. Young TL, Deeb SS, Ronan SM, et al, X-linked high myopia associated with cone dysfunction, *Arch Ophthalmol*, 122(2004,)897-908
  57. Young T.L, Ronan S.M, Drahozat L.A, Wildenberg SC, Alvear AB, et al. Evidence that a locus for familial high myopia maps to chromosome 18p. *Am J Hum Genet*, 63;( 1998) 109-119
  58. Young T.L, Ronan S.M, Alvear AB, Wildenberg SC, Ocuing WS, et al. A second locus for familial high myopia maps to chromosome 12q. *Am J Hum Genet*, 63 (1998)1419-1424
  59. Prasuna Paluru, Shawn M. Ronan, Elise M. Heon, et al. New locus for autosomal dominant high myopia maps to the long arm of chromosome 17, *Invest Ophthalmol Vis Sci*, 44(5) (, 2003) 1830-1836
  60. Naiglin L, Gazagne Ch, Dallongeville F, Thalamas C, Idder A, et al. A genome wide scan for familial high myopia suggests a novel locus on chromosome 7q36, *J Med Genet*, 39(2002)118-124
  61. Prasuna Paluru, Nallasamy S, Devoto M, Rappaport EF & Young TL, Identification of novel locus on 2q for autosomal dominant high-grade myopia, *Invest Ophthalmol Vis Sci*, 46(2005)2300-2307
  62. Lam DSC, Tam POS, Fan DSP, Baum L, Leung Y-F, Pang CP, Familial high myopia linkage to chromosome 18p,

- Ophthalmologica*, 217 (2003) 115-118
63. Mutti DO, Semina E, Marazita M, Cooper M, Murray JC, et al, Genetic loci for pathological myopia are not associated with juvenile myopia, *Am J Med Genet*, 112 (2002) 355-360
  64. Scavello GS, Paluru PC, Zhou J, White PS, Rappaport EF, et al, Genomic structure and organization of the high grade myopia-2 locus (MYP2) critical region: mutation screening of 9 positional candidate genes, *Mol Vis*, 11 (2005) 97-110
  65. Paluru PC, Scavello GS, Ganter WR & Young TL, Exclusion of lumican and fibromodulin as candidate genes in MYP3 linked high grade myopia, *Mol Vis*, 10 (2004) 917-922
  66. Lam DSC, Lee WS, Leung YF, Tam POS, Fan DSP, et al, TGF  $\alpha$ -induced factor: A candidate gene for high myopia, *Invest Ophthalmol Vis Sci*, 44 (2003) 1012-1015
  67. D. Lo RS, Lee S, Massague J, A Smad transcriptional corepressor, *Cell*, 97 (1999) 29-39
  68. Watton D, Lo RS, Awaby LAC & Massague J, Multiple modes of repression by the Smad transcriptional corepressor TGIF, *J Biol Chem*, 274 (1999) 37105-37110
  69. Bertolino E, Reimund B, Wildt-Perinic D, Clere RG, A novel homeobox protein which recognizes TGT core and function interferes with a retinoid- responsive motif *J Biol Chem*, 270 (1995) 31178-31188
  70. Overhauser J, Mitchell HF, Zackai EH, Tick DB, Rojas K & Muenke M, Physical mapping of the holoprosencephaly critical region in 19p11.3, *Am J Hum Genet*, 5 (1995) 1080-1085.
  71. Muenke M & Beachy PA. Genetics of ventral forebrain development and holoprosencephaly. *Curr Opin Genet Dev*, 10 (2000) 262-269
  72. Gripp KW, Wotton D, Edwards MC, Roessler E, Ades L, et al., Mutations in TGIF cause holoprosencephaly and link NODAL signaling to human neural axis determination, *Nat Genet*, 25 (2000) 205-208
  73. Chen C. P, Chen S. R, Du S. H & Wang W. Molecular diagnosis of a novel heterozygous 268C3T(R90C) mutation in TGIF gene in a fetus with holoprosencephaly and premaxillary agenesis, *Prenat Diagn*, 22 (2002) 5-7

# Inflammation and atherosclerosis

Azra Shah, Yasmeen Rauf,

Over the last few decades it has become increasingly clear that atherosclerosis is not merely a disease caused by increased accumulation of lipids in the arterial walls but a more complex process, the nature of which is inflammatory. Inflammation is involved in the initiation (atherogenesis), lesion progression and ultimately the complications (Plaque disruption and thrombosis) of atherosclerosis. Most of this evidence has come from clinical and epidemiologic data as from basic morphologic studies<sup>1-3</sup>.

## Atherogenesis:

The chronic inflammatory response of the arterial wall is initiated by some form of chronic or repetitive injury to the endothelium<sup>4</sup>. Animal studies have shown that initiation with an atherogenic diet causes changes in the endothelial cells with development of focal regions of chronic endothelial injury usually subtle with resultant endothelial dysfunction leading to increased endothelial permeability and increased leukocyte adhesion<sup>5</sup>. Early human lesions may however, develop at sites of morphologically intact endothelium<sup>6</sup>. Thus non-denuding endothelial dysfunction and activation are more important in human disease; these are manifested by increased endothelial permeability, increased leucocyte adhesion and alterations in expression of a number of endothelial gene products like intracellular adhesion molecule 1 (ICAM-1) and vascular adhesion molecule 1 (VCAM-1) leading to adhesion of blood monocytes and T cells to the endothelium with migration of blood monocytes into intima and their transformation into foam cells and macrophages<sup>7-9</sup>. This migration is mediated by chemokines like mono-attractant protein 1 (MCP-1) generated by the macrophages<sup>10</sup>.

The process after initiation is augmented by expression of inflammatory cytokines such as macrophage colony stimulating factor (M-CSF) which modify macrophages so that they ingest more lipoprotein particles and promote macrophage proliferation. This process is augmented by attraction and participation of B and T cells. Later cytokines (L – interferon and tumour necrosis factor alpha (TNF $\alpha$ )) are secreted to cause migration and proliferation of smooth muscle cells (SMCs) and stimulation of endothelial cells<sup>11</sup>. The inflammation further causes the SMCs to secrete enzymes which digest and degrade structural proteins in the arterial wall allowing migration of SMCs out of the media into the intima thereby initiating plaque development<sup>12</sup>. This migration is also caused by release of factors from platelets (platelet derived growth factor PDGF) and vascular cells. The SMCs proliferate in the intima, elaborating intracellular matrix leading to accumulation of collagen, elastin and proteoglycans thus converting the fatty streak into a fibrofatty atheroma<sup>13</sup>.

What initiate endothelial dysfunction in early atherosclerosis? Endotoxins, hypoxia, products of cigarette smoke, homocysteine, viruses and other infectious agents have been implicated<sup>14</sup>. The two most important determinants of endothelial alteration are hemodynamic disturbances and adverse effects of hypercholesterolemia<sup>15,16</sup>.

## Plaque Progression:

On continuation of the inflammatory process more mediators are released which promote SMC proliferation. The CD 40/CD 40L interaction between activated T cells and macrophages leads to expression of tissue factor (TF) which promotes coagulation, expression of metalloproteinases (MMPs) promoting protein degradation and expression of additional pro-inflammatory cytokines<sup>17</sup>. Mast cell degranulation also causes plaque progression. Continuation of the inflammatory process leads to progression of lesion to complex plaques with necrotic lipid cores and neovascularization. Rupture of the fragile vessels leads to plaque expansion and increase in the lipid core leads to plaque instability. Pro-inflammatory

## Authors affiliations:

**Prof. Azra Shah**

SKIMS, Soura  
Srinagar Kashmir India

**Yasmeen Rauf**

SMHS, Hospital

**Accepted for Publication**

December 2006

## Correspondence:

**Prof. Azra Shah**

Head Department of Pathology  
SKIMS, Soura Srinagar

**JK-Practitioner 2007;14(2):71-74**

cytokines secreted by activated T cells decrease production of structural proteins that help stabilize the plaque with death of SMCs and macrophages leading to decreased collagen synthesis. MMPs lead to degradation of fibrous cap and these events ultimately cause plaque vulnerability and weakness and thrombotic tendencies<sup>137</sup>.

#### **Vulnerable Plaques:**

It has been shown that majority of acute coronary events occur at arterial sites which do not show significant stenosis but rather have plaque characteristics that make them prone to thrombosis. The plaques occur at branch points and along posterior wall of the descending and abdominal aorta, supporting the concept of hemodynamic disturbances. The turbulent flow and shear stress cause focal areas of endothelial dysfunction and predispose to development of lesions at these sites<sup>138</sup>. Alterations of shear stress and disturbed flow induce endothelial genes with potential pro-inflammatory and proatherogenic activities including cytokines and adhesion molecules. The products of these genes actually protect the lesion and these are therefore, named atheroprotective genes<sup>139</sup>. This explains the non-random localization of early atherosclerotic lesions. This also causes increased endothelial permeability and cell turnover and enhanced receptor mediated low density lipoprotein (LDL) endocytosis and insulation into the vessel wall and modification by oxidation<sup>140</sup>. The factors which promote plaque instability are circumferential and shear stress, vasospasm, necrotic lipid core, neovascularization, erosion and fissuring of fibrous cap, MMPs, SMCs, nodular calcification and inflammation<sup>141</sup>.

The main initiating factor of thrombosis is plaque erosion and fissuring which exposes the prothrombotic surface to the luminal blood causing adhesion of platelets to these focal points of endothelial denudation and to the adherent leukocytes. Release of factors from the activated macrophages, platelets or vascular cells cause migration of SMCs from the media to the intima. In case of plaque fissuring the process is more dramatic and subsurface elements like collagen, TF, Von Willebrand factor (VWF) are exposed. Other factors like interleukin 1 (IL1), tumour necrosis factor alpha (TNF $\alpha$ ) and CD 40 L cause further adhesion of leukocytes<sup>142</sup>.

#### **Smooth muscle cell proliferation:**

If the inciting event persists, SMCs proliferate, extra-cellular matrix is deposited in the intima and the fatty streak is converted into a fibro-fatty atheroma leading to progression of the atherosclerotic lesions<sup>143</sup>. The SMC proliferation is modulated by inhibitors including heparin like molecules present in the endothelial cells and SMCs or TGF-B derived from endothelial cells and macrophages. Further more activated inflammatory cells and immune cells in the plaque can lead to death of intimal SMCs by apoptosis<sup>144</sup>.

#### **Role of inflammation in atherosclerotic risk factors:**

**Role of lipids:** Hypercholesterolemia impairs endothelial function. Chronic lipidemia causes accumulation of lipids within the intima at sites of increased endothelial permeability. Oxidative modification of lipids by free radicals generated in macrophages or endothelial cells yields oxidized LDL (ox LDL)<sup>145</sup>.

These modified lipoproteins induce inflammatory

changes in the arterial wall. ox LDL promotes expression of adhesion molecules and induces chemokines and pro-inflammatory cytokines. The apoprotein part acts as an antigen for T cells thereby inducing an inflammatory response as well. This is chemotactic for circulating monocytes and decreases the motility of macrophages. It is also immunogenic inducing production of antibody to ox-LDL. Very low density lipoproteins (VLDL) and other lipoproteins have significant atherogenic potential, they are oxidized to produce superoxide and other free oxygen radicals that deactivate nitric oxide, the major endothelium-releasing factor. Oxidative stress also activates NF LB and endothelial gene expression of numerous biologically active molecules. High density lipoproteins (HDL) may have the opposite effect<sup>146</sup>. This concept of hyperlipidemia and oxidative stress is consistent with experimental and clinical studies showing protection from atherosclerosis by antioxidant vitamins ( $\beta$  Carotene and Vitamin E) and drugs that decrease generation of oxidants (probucol)<sup>147</sup>. Moreover, cholesterol lowering drugs and antioxidants therapy improve clinical measures of endothelial dysfunction<sup>148</sup>. Further work is being done to elucidate the role of oxidative stress in atherosclerosis.

**Hypertension** causes increased stress on the arterial wall leading to endothelial dysfunction. It also has a link with inflammation through angiotension II which has been implicated in the production of oxygen species and promotion of effects of inflammatory cytokines IL-6 and MCP-1.

**Diabetes Mellitus** through hyperglycemia causes changes in the macromolecules with formation of advanced glycation end products (AGE). These proteins interact with receptors and cause expression of pro-inflammatory cytokines<sup>149</sup>.

**Obesity** leads to diabetes mellitus through insulin resistance. It also causes elevation of lipoproteins associated with atherogenesis. The adipose tissue also produces cytokines such as IL-6 and TNF  $\alpha$ <sup>150</sup>.

#### **Role of infectious agents:**

Generalized infections can lead to the production of circulating inflammatory cytokines and mediators which influence the promotion of atherosclerotic lesions<sup>151</sup>. "In animals certain viruses like Mareks disease agent cause plaques in the aorta" and also lead to vasculitis<sup>152</sup>. In addition, coagulation, fibrinolysis and hemodynamics are all influenced by general and chronic infections<sup>153</sup>. Herpes virus and cytomeglo virus (CMV) have been detected in human atherosclerotic plaques<sup>154</sup>. Epidemiological and therapeutic studies seeking the association between atherosclerosis and organisms like chlamydia pneumonia, Helicobacter pylori, herpes, simplex virus and CMV have however remained elusive. Antibiotic therapy has however, shown to reduce recurrent clinical events in patients with ischemic heart disease. Gupta et al<sup>155</sup> have shown that infective organisms might potentiate the complications of existing lesions. It may also potentiate the local effects of known risk factors like hypercholesterolemia by accelerating the chronic inflammatory pathways or altering the response of vascular wall to injury through circulating inflammatory

mediators<sup>11,12</sup>.

### **Inflammatory makers in atherosclerosis:**

New markers for cardiovascular risk and development of atherosclerosis have recently come up after the role of inflammation in promoting heart disease has been explained<sup>13,14</sup>. A recent publication stresses on "vulnerable patient" rather than a "vulnerable plaque". The vulnerable patient includes the triad of vulnerable plaque, vulnerable blood and vulnerable myocardium<sup>15</sup>. A significant correlation between the inflammatory marker C reactive protein (CRP), IL-6 and TNF $\alpha$  in older patients with cardiovascular events has been shown by the Health ABC study (2003)<sup>16</sup> carried out on 2200 patients. These markers were independent precursors of cardiovascular events in the elderly. Furthermore, levels of CD 40L in the blood in healthy women and in patients with acute coronary syndromes showed an increase in future cardiovascular events<sup>17</sup>. This study points out towards the role of these markers in the pathogenesis of atherosclerosis.

### **CRP:**

This was the first marker to be associated with atherosclerosis on observation that its high levels were an independent prediction of cardiac events, some studies showed it to be a better predictor than LDL<sup>18,19</sup>. It causes better endothelial cell activation, survival and proliferation of SMCs indicating it to be a powerful proatherosclerotic factor.

**Interleukin 18** is a member of the interleukin family and has strong association with coronary artery disease. It plays a role in the inflammatory component of atherosclerosis. Its blockade leads to formation of a stable plaque with lesser inflammation and lipids, increased collagen and SMCs. Its levels have also shown to decrease with weight loss<sup>20</sup>.

### **CD 40/CD 40L:**

This system was originally described in T and B cell interaction and recently has been shown to be involved in the pathogenesis of chronic inflammatory diseases including atherosclerosis. It is expressed by T cells, endothelial cells, SMCs and macrophages. Its changes ultimately lead to plaque instability and plaque vulnerability due to non-repair of vascular erosions. This is a good predictor of serious and fatal complications in women with acute coronary artery disease<sup>21</sup>.

### **Protease-activated receptors (PARs)**

These are involved in tissue responses like inflammation and repair, in the initiation, progression and

complications of atherosclerosis. They play a role in expressing cytokines, causing inflammatory cell adhesion, SMC migration and platelet activation<sup>22,23</sup>.

### **Lectin like oxidized LDL receptor 1(LOX1):**

This has been identified as a major receptor for ox LDL in endothelial cells, macrophages and SMCs. In addition to binding ox LDL, it also binds apoptotic cells, platelets, AGE products and pathogenic organisms. It potentiates atherogenesis by decreasing nitric oxide production, inducing apoptosis, causing monocyte adhesion and triggering the CD 40/CD 40L path way. Reactive oxygen species (ROS) are produced which inactivate NO and lead to instability of the fibrous cap<sup>24</sup>.

### **Lipoprotein – associated phospholipase A2:**

(Lp-PLA2) and secretory phospholipase A2 (SPLA2)

Plasma levels of Lp-PLA2 are a strong risk factor for coronary artery disease (CAD) in patients with hypercholesterolemia but not in healthy patients. This enzyme produces two products, lysophosphatidyl choline (Lyso PC) and non-esterified fatty acid compounds. The former attracts monocytes. Plasma levels of SPL A2, an acute phase reactant have been shown to be a new independent risk factor for CAD<sup>25</sup>.

### **Matrix Metaloproteinase-9 (MMP-9):**

This is a family of enzymes containing zinc and are involved in the proteolysis of extra-cellular proteins like collagen, elastin and proteoglycans. The destruction of elastic fibers contributes to formation of atherosclerotic aneurysms<sup>26</sup>. These enzymes are inhibited by tissue inhibitors of metallo-proteinases (TIMPs). They play a role in vascular remodeling, restenosis, cardiac dysfunction and atherosclerosis. These are found in the shoulder region of atherosclerotic plaques, which is the commonest site of plaque disruption. Elevated plasma levels have been shown in type II diabetic patients with CAD. Elevated levels have been shown to have an increased risk of death in heart disease patients. Statins inhibit their secretion thus leading to plaque stabilization<sup>27</sup>.

These new markers are exciting developments in the understanding of atherogenesis and in the development of its complications. They also allow us to study the role of inflammation and to identify additional groups of patients at risk for heart disease and to identify new avenues for pharmacological intervention independent of or in addition to lipid modulation.

## **References**

- Ross R. The pathogenesis of atherosclerosis, a perspective for the 1990s. *Nature* 1993;362:801-09.
- Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. *Circulation* 2001;101:2266.
- Cybulsky MI, Gimbrone MA. Endothelial expression of a mononuclear leucocyte adhesion molecule during atherogenesis. *Science* 1991;251:788.
- Cotran RS. New roles for the endothelium in inflammation and immunity. *Am J Pathol* 1987;129:407.
- Ross R. Atherosclerosis – an inflammatory disease. *New Eng J Med* 1999;340:115.
- Libby P. Inflammation and atherosclerosis. *Circulation* 2002;105:1135.
- O Brein KD. Neovascular expression of E-selection, intercellular adhesion molecule I and VCAM I in human atherosclerosis and their relation to intimal leucocyte content. *Circulation* 1996;93:672.
- Gimbrone MA, Nagel T, Topper JN. Bio-mechanical activation, an emerging paradigm in endothelial adhesion biology. *J Clin Invest* 1997;99:1809.
- Gimbrone MA Jr, Topper JN, Nagel T, Anderson KR et al. Endothelial dysfunction, hemodynamic forces and atherogenesis. *Ann NY Acad Sci* 2002;902:230-39.
- Tousoulis D. Inflammatory and thrombotic mechanisms in coronary atherosclerosis. *Heart* 2003;89:993.
- Young JL, Libby P, Schonbeck U. Cytokines in the pathogenesis of

- atherosclerosis. *Thromb Haem* 2002;88:554.
12. Raines EW, Ross R. Smooth muscle cells and the pathogenesis of the lesions of atherosclerosis. *Br Heart J* 1993;69:S30-S37.
13. Stary HC, Chandler AB, Glagov HS, Guyton JR et al. A definition of initial fatty streak and intermediate lesions of atherosclerosis. A report from the committee on vascular lesion of the council of atherosclerosis. *Arterioscler. Throm* 1994;14:840-56.
14. Schoen FJ. In Robbins and Cotran. *Pathologic basis of disease*. Eds Kumar V, Abbas AK, Fausto N. 7<sup>th</sup> ed Saunders, Philadelphia 2004;511-554.
15. Gimbrone MA Jr. Vascular endothelium, hemodynamic forces and atherogenesis. *Am J Pathol* 1999;155:1-5.
16. Gimbrone MA Jr. Endothelial dysfunction, hemodynamic forces and atherogenesis. *Ann NY Acad Sci* 2001;947:93.
- 16a. Berk BC. Endothelial atheroprotective and anti-inflammatory mechanisms. *Ann NY Acad Sci* 2000;902:239.
17. Schonbeck U. Soluble CD 4DL and cardiovascular risk in women. *Circulation* 2003;108:2113.
18. Libby P. Inflammation in atherosclerosis. *Nature* 2002;420:848.
19. Libby P. Vascular biology of atherosclerosis. Overview and state of the art. *Am J Cardiol* 2003;91:3A.
20. Ross R. Atherosclerosis. Current understanding of mechanisms and future strategies in therapy. *Transpl Proc* 1993;25:2041-43.
21. Ross R. Whipple award lecture. Atherosclerosis. A defense mechanism gone awry. *Am J Pathol* 1993;143:982-1002.
22. Hort W. Arteriosclerosis. Its morphology in the past and today. *Basic Res Cardiol* 1994;89:1-15.
23. Dickson BC, Gotlieb AI. Towards understanding acute destabilization of vulnerable plaques. *Cardiovasc Pathol* 2003;12:237.
24. Cornhill JF, Roach MR. A quantitative study of the localization of atherosclerotic lesions in rabbit aorta. *Atherosclerosis* 1976;23:489.
25. Zarins CK, Giddens DP, Bhairdvarj BK et al. Carotid bifurcation atherosclerosis. Quantitation of plaque localization with flow velocity profiles and wall shear stress. *Circ Res* 1983;53:502.
26. Geng YJ, Libby P. Progression of atheroma, a struggle between life and procreation. *Arterioscler Thromb Vasc Biol* 2002;22:1370.
27. Steinberg D. Oxidative modification of low density lipoprotein and atherogenesis. *Circulation* 1977;95:1062.
28. Treasure CB. Beneficial effects of cholesterol lowering therapy on the coronary endothelium in patients with coronary artery. *New Eng J Med* 1995;332:481.
29. Anderson TJ et al. The effect of cholesterol lowering and antioxidant therapy on endothelium dependent coronary vasomotion. *New Eng J Med* 1995;332:488.
30. Shimizu K. Direct anti inflammatory mechanisms contribute to attenuation of experimental allograft atherosclerosis by statins. *Circulation* 2003;108:2113.
31. Beckman JA, Creagar MA, Libby P. Diabetes and atherosclerosis, epidemiology, Patho-physiology and management. *JAMA* 2002;287:2570.
32. O Connor S. Potential infectious etiologies of atherosclerosis, a multifactorial perspective. *Emerg Inf Dis* 2001;7:780.
33. Streblow DN. Do pathogens accelerate atherosclerosis? *J Nutr* 2001;131:2798S.
34. Kalayoglu MV, Libby P, Byrne GI. C pneumonia as an emerging risk factor in cardiovascular disease. *JAMA* 2002;288.
35. Fabricant CG, Fabricant J, Litrenta MM, Minick CR. Virus induced atherosclerosis. *J Exp Med* 1978;148:335.
36. Week KE. Murine herpes virus 68 causes severe large vessel arteritis in mice lacking interferon alpha responsiveness: a new model for virus-induced vascular disease. *Nat Med* 1997;3:1346.
37. Biyu LM. Does atherosclerosis have an infectious etiology. *Circulation* 1996;94:872.
38. Cunningham MJ, Pasternak RC. The potential role of viruses in the pathogenesis of atherosclerosis. *Circulation* 1988;77:964.
39. Gupta S. Elevated C pneumonia antibodies, cardiovascular events and azithromycin in male survivors of myocardial infarction. *Circulation* 1997;96:404.
40. Libby P. Roles of infectious agents in atherosclerosis and restenosis: an assessment of the evidence and need for further research. *Circulation* 1997;96:4095.
41. Rosenson RS, Koenig W. Utility of inflammatory markers in the management of coronary artery disease. *Am J Cardiol* 2003;92:10.
42. Szmítko PE. New markers of inflammation and endothelial activation Part I. *Circulation* 2003;108:1917.
43. Szmítko PE. Biomarkers of vascular disease linking inflammation to endothelial activation. Part II. *Circulation* 2003;108:2041.
44. Naghavi N. From vulnerable plaque to vulnerable patient. A call for new definitions and risk assessment strategies Parts I & II. *Circulation* 2003;108:1664 and 1772.
45. Cesari M. Inflammatory markers and onset of cardiovascular events. Results from the Health ABC study. *Circulation* 2003;108:2317.
46. Varo N. Soluble CD 40L. Risk prediction after acute coronary syndromes. *Circulation* 2003;108:1049.
47. Griending KK, Fitzgerald GA. Oxidative stress and cardiovascular injury. Part I. Basic mechanisms and in vivo monitoring of ROS. *Circulation* 2003;108:1912. Part II. Animal and human studies. *Circulation* 2004;108:2034.
48. Burke A, Virmani R. Heart and blood vessels in Modern Surgical Pathology Vol I. Eds. Weidner N, Cote RJ, Suster S and Weiss LM. Saunders, Philadelphia 2003;505-35.
49. Nakashima Y, Kurozumi T, Sueishi K, Tanaka K. Dissecting aneurysm, a clinico-pathologic and histopathologic study of 111 autopsied cases. *Hum Pathol* 1990;21:291-96.

# Clinical pattern and etiology of pleural effusion: Experience from Riyadh Medical Complex

Abdullah A. Abba, Mohamed Z. Khalil, Fahad Al-Majeed

## Abstract

This study was a retrospective analysis of data obtained from the medical records of patients admitted to a 1500 bed community hospital in central Saudi Arabia with a diagnosis of pleural effusion over a one year period between January 2005 and December 2005. Demographic, clinical, laboratory and radiographic data were extracted. Final diagnosis was based on compatible clinical and laboratory features and response to treatment. A total of 107 patients with mean age of  $48.7 \pm 18.9$  years (range 13-90), with a diagnosis of pleural effusion were included in the study. Seventy three patients (68.2%) were males and 68 (63.6%) were Saudi. The etiology was established in 101 patients (94.4%) patients. The most common presenting clinical features were productive cough and breathlessness in 23 patients (21.4%) followed by fever in 20.5% of patients. Patients presenting with tuberculous pleural effusion were significantly younger than those with cardiac failure and malignancy ( $p < 0.007$ ). The pleural effusion was exudative in 76 (71%) patients. Parapneumonic effusion was found in 33 (30.8%) patients leading other causes, while CCF, malignancy, and TB were less commonly found in 24 (22.4%), 14 (13.1%), and 12 (11.2%) patients respectively ( $p < 0.0001$ ). The duration of stay was not significantly different between the groups but patients with complicated parapneumonic effusions stayed significantly longer than patients with simple effusions ( $p < 0.001$ ). The clinical presentation of pleural effusion is variable; however, productive cough and dyspnea are the most common symptoms. The rank order of causes of pleural effusion in our setting seems to vary from other local studies and studies from other countries. This may be a reflection of the changing disease pattern in the Kingdom.

*JK-Practitioner 2007;14(2): 75-78*

## Introduction:

Pleural effusion is an excessive accumulation of fluid between the parietal and visceral pleural layers resulting from local disease or a systemic cause of an alteration in the haemodynamic factors governing the clearance of pleural fluid. It is a common problem caused by a variety of mechanisms and diseases. Despite the advances made in diagnostics, the etiology cannot be determined in some patients with pleural effusion. This figure may be as high as 25% of patients. It is imperative to establish the diagnosis of pleural effusion in every patient, and every attempt should be made to reach a correct diagnosis. Pleural effusion indicates the presence of disease that may be pulmonary, pleural, or extrapulmonary. The standard approach would be a thorough history and physical examination aided by diagnostic evaluation. While several guidelines are provided to help clinician in their work up for pleural effusion, among which is the British Thoracic Society guidelines for the investigation of a unilateral pleural effusion in adults, knowledge of the local pattern definitely aids in streamlining the approach. The type of subject covered by a study also determines the etiologic pattern.

In the Kingdom of Saudi Arabia (KSA), the available data on the etiology and clinical pattern of pleural effusions are not sufficient to draw a conclusion. Furthermore, the etiology of pleural effusion may be changing due to the rapid urbanization and hence the change in disease pattern affecting the people living in KSA. Therefore, we designed and conducted this study to obtain the clinical pattern and etiology of pleural effusions among adults admitted to a large general hospital in KSA.

## Materials and Methods:

This is a hospital based retrospective study, conducted by acquiring data from the medical records of patients admitted to a large community hospital of 1500 beds in Central Saudi Arabia with a diagnosis of pleural effusion over one year period (between January 2005 and December 2005). All patients admitted with a primary or secondary diagnosis of pleural effusion according to the ICD classification (511.9) were included. Demographic, clinical, laboratory and radiological features were documented. Pleural fluid analysis was used to classify effusions into exudates or transudates based on Light's criteria. Treatment and outcome

## Authors affiliations:

**Abdullah A. Abba, Mohamed Z. Khalil, Fahad Al-Majeed**

Department of Medicine, King Khalid University Hospital and College of Medicine, King Saud University Riyadh Saudi Arabia

## Accepted for Publication

November 2006

## Correspondence:

**Dr. Abdullah A. Abba**

Consultant & A. Professor of Medicine

King Khalid University Hospital & College of Medicine

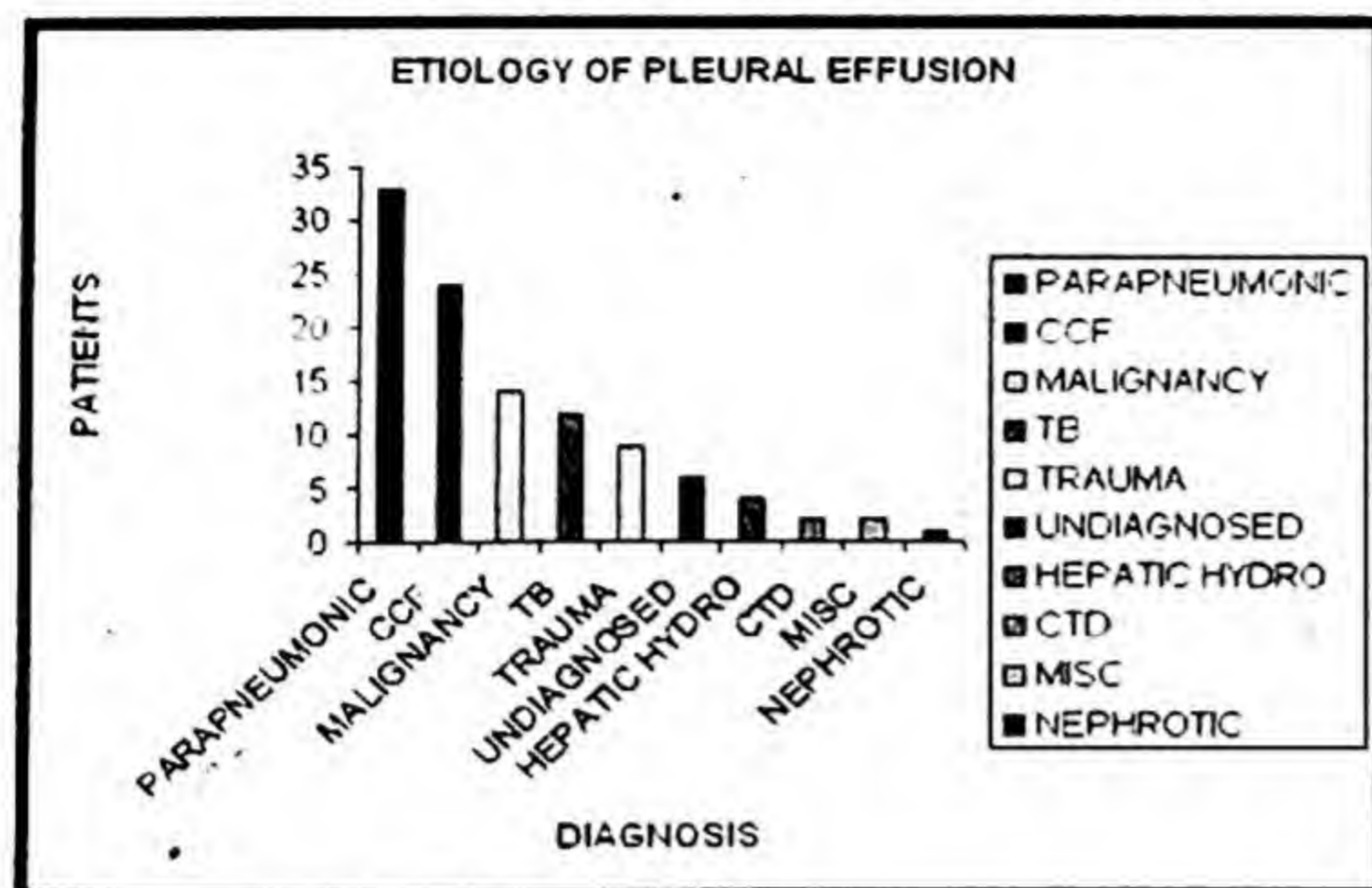
P.O.Box. 50726,

Riyadh 11533,

Saudi Arabia

Keywords : pleural effusion-etiology-Saudi Arabia-TB-parapneumonic

Fig:1 Etiology of Pleural effusion in order of frequency



were noted. The final diagnosis was based on compatible clinical and histological features and therapeutic response based on the following criteria: parapneumonic effusion – underlying pneumonia, bronchiectasis or lung abscess or presence of purulent fluid or positive bacterial culture from fluid; congestive heart failure – compatible clinical features, absence of lung infiltrates and response to antifailure therapy; tuberculous – presence of acid fast bacilli in fluid or pleural biopsy or caseating granuloma on pleural histology with adequate response to anti-tuberculous therapy; neoplastic – presence of neoplastic cells on pleural cytology or histology. Clinical features and relevant investigations were used to classify other miscellaneous causes. It was classified as idiopathic where there were no compatible clinical features and two pleural biopsies were not diagnostic. Statistical analysis was done using the SPSS program with a level of significance at 5%.

positive bacterial culture from fluid; congestive heart failure – compatible clinical features, absence of lung infiltrates and response to antifailure therapy; tuberculous – presence of acid fast bacilli in fluid or pleural biopsy or caseating granuloma on pleural histology with adequate response to anti-tuberculous therapy; neoplastic – presence of neoplastic cells on pleural cytology or histology. Clinical features and relevant investigations were used to classify other miscellaneous causes. It was classified as idiopathic where there were no compatible clinical features and two pleural biopsies were not diagnostic. Statistical analysis was done using the SPSS program with a level of significance at 5%.

Results: One hundred and twenty six patients were classified with a primary or secondary diagnosis of pleural

effusion during the period of study. Complete data could be retrieved from 107 of the charts. The average age of the population was 48.69 years  $\pm$  18.89 (range 13 – 90). There were 73 (68.2%) males and 68 (63.6%) Saudi. Seventy seven (71.9%) of the patients had exudative effusions according to Light's criteria while 30(28.1%) had transudates. Figure 1 gives the etiology of pleural effusion, the most common being parapneumonic effusion 33(30.8%), heart failure 24(22.4%), malignancy 14(13.5%) and tuberculosis 12 (11.2%). Others include trauma 9(8.4%), hepatic hydrothorax 4(3.7%), connective tissue disease 2(1.9%) and nephrotic syndrome in 1(0.9%). In 6(5.6%) patients there was no definite diagnosis in spite of extensive investigations. Table 1 shows that tuberculous effusion and trauma is significantly more common among a younger group of patients. A one way analysis of variance between the etiological factors and age showed significantly younger population had tuberculosis than heart failure ( $p < 0.0001$ ) and malignancy ( $p < 0.007$ ). Productive cough and dyspnoea were the most commonly encountered symptoms irrespective of the etiology. It is clear that there is no clear symptom pattern that favors a particular etiology although a complaint of lower limb

Table 1: Distribution according to nationality gender, age and duration of stay among the common causes of pleural effusion:

DIAGNOSIS	NATIONALITY		GENDER		AGE(yrs) Mean(SD)	DURATION O: STAY(days) Median(SD) -
	Saudi (%)	Non-Saudi (%)	Male(%)	Female(%)		
Parapneumonic	25 (75.8)	8 (29.2)	22 (66.7)	11 (33.3)	45.64 (+/-20.13)	14 (+/-22.7)
CCF	16 (66.7)	8 (33.3)	18 (75)	6 (25)	66.63 (+/-10.42)	8 (+/-7.4)
Malignancy	9 (64.2)	5 (35.8)	8 (52.1)	6 (42.9)	56.45 (+/-19.16)	19.5 (+/- 13)
Tuberculosis	5 (41.7)	7 (58.3)	8 (66.7)	4 (33.3)	31.33 (+/-13.20)	14 (+/-8.1)
Trauma	5 (55.6)	4 (44.4)	8 (88.9)	1 (11.1)	37.7 (+/-14.68)	18 (+/- 9.4)
Hepatic	3 (75)	1 (25)	2 (50)	2 (50)	52.5 (+/- 18.41)	17 (+/-9.3)
Miscellaneous (including CTD,	3(60)	2(40)	4(80)	1(20)	37.40 (+/-16.86)	13 (+/- 8.1)
Undiagnosed	4(66.7)	2(33.3)	3(50)	3(50)	48.5 (+/-17.86)	16 (+/-4.9)

edema is most often found in patients with heart failure. Effusion occurred on the right side in 53(49.5%), left in 36(33.6%) and bilateral in 18(16.9%) patients. Table 2 gives the side of effusion in relation to the etiology showing that bilateral pleural effusion was almost always due to cardiac failure. All patients with PPE received appropriate antibiotics with 9(27.3%) patients requiring intercostal tube drainage with or without fibrinolytic agent and 3 (9.1%) patients required surgical decortication. This group of patients spent a median of 25 days in hospital as opposed to

the median duration of stay of 14 days for the group of patients with parapneumonic effusions ( $p < 0.001$ ). During the period of study 9 patients (8.4%) died – three with associated human immunodeficiency virus infection, four with metastatic carcinoma and another two with congestive cardiac failure.

#### Discussion:

Pleural effusion remains a commonly encountered clinical problem with considerable difficulty in reaching the underlying etiology. The data obtained from our study are clearly demonstrating that parapneumonic (PPE) effusion is the most common cause of pleural effusion among our study population. The change in pattern of pleural effusion to be commonly due to PPE as observed in our study may be explained by either a true change in the etiology of pleural effusion, or selection bias as our hospital is a general hospital, and most patients with TB would have been referred to a nearby hospital TB hospital. Alternatively, we may suggest under-treatment of pneumonia leading to the development of PPE or rather increasing prevalence of more virulent organisms that eventually lead to PPE. A recent study by Light reported that parapneumonic effusions occur in 20 to 40% of patients who are hospitalized with pneumonia<sup>10</sup>. The prognostic significance of this finding is rather important, as the infection-related mortality rate of complicated parapneumonic effusions patients was shown by one study to reach 41%<sup>11</sup>.

Furthermore, PPE are associated with prolonged hospital stay due to several factors. In a recent study, factors influencing length of hospital stay in PPE have been identified as follows: purulent pleural fluid, underlying disease, surgical drainage with or without decortication, higher pleural fluid levels of lactate dehydrogenase and polymorphonuclear elastase<sup>12</sup>. The median duration of stay in patients with any of these factors was found, in that study, to be 17 days. Our patients had a more prolonged stay with the added cost implications. This further emphasizes the need for the immediate evaluation and prompt management of parapneumonic effusions as stressed by Light and Sahn<sup>13</sup>. Patients with pleural effusions with the following characteristics would generally need some form of invasive procedure: effusions exceeding 50% of thoracic space, purulent or loculated, positive Gram's stain or culture, pH of 7.2, glucose 60mg/dL or lactate dehydrogenase level of more than 3 times the upper limit of normal. This may range from simple aspiration to the more traumatic decortication. An earlier study has noted excessive mortality in patients with PPE<sup>14</sup>. Although no mortality has been recorded in our series, a significant number of our patients had to undergo invasive procedures thus prolonging their stay and increasing healthcare costs. Selection bias notwithstanding, the possibility of under treatment of community acquired pneumonia or the development of more virulent organisms leading to PPE requiring invasive procedures and hence prolonged hospital stay, calls for special awareness of this complication and prompt action.

It is of no surprise to find congestive cardiac failure (CCF) as the second most common underlying etiology of pleural effusion in our study population,

Table 2. Side of pleural effusion in relation to etiology

DIAGNOSIS	RIGHT	LEFT	BILATERAL	TOTAL
Parapneumonia	20 (60.6%)	12 (36.4%)	1 (30%)	33
Heart Failure	7 (29.2%)	4 (16.7%)	13 (54.1%)	24
Malignancy	8 (57.2%)	5 (35.7%)	1 (7.1%)	14
Tuberculosis	5 (41.7%)	6 (50%)	1 (8.3%)	12
Trauma	4 (44.4%)	4 (44.4%)	1 (11.2%)	9
Hepatic	3 (75%)	1 (25%)	-	4
Nephrotic	1 (100%)	-	-	1
Miscellaneous	1 (25%)	2 (50%)	1 (25%)	4
Undiagnosed	4 (66.7%)	2 (33.3%)	-	6
Total	53 (49.5%)	36 (33.6%)	18 (16.9%)	107

particularly, with the increasing prevalence of heart failure. Recent studies from the Kingdom indicate high prevalence of diabetes mellitus and metabolic syndrome – all potential underlying predisposing factors of coronary artery disease and hence heart failure<sup>15</sup>.

Previous studies in the kingdom have shown tuberculosis to be the leading cause of pleural effusion comparable to other areas of high incidence of tuberculosis<sup>16</sup>. It has been shown that the annual incidence rates of extrapulmonary tuberculosis have been increasing over the last few years in the Kingdom of Saudi Arabia, and true rates may even be higher due to incomplete reporting<sup>17</sup>. A recent study of the prevalence of respiratory diseases in hospitalized patients in Saudi Arabia has however ranked tuberculosis (7.2%) after both pneumonia (11.5%) and lung cancer (8.4%) suggesting our finding to be a genuine reflection of the changing pattern of the disease in the Kingdom<sup>18</sup>.

We found that productive cough, dyspnea, and fever were the most common presenting clinical features of our patients. The pattern of symptoms was not helpful in the elucidation of the etiology of pleural effusion. Pleural effusion was commonly found on right side (44.9%), compared to left side (34.6%) or bilateral (20.5%) and this was statistically significant ( $p < 0.001$ ). These observations were in keeping with previous reports<sup>19</sup>. The presence of bilateral effusions in the setting of compatible symptoms however, necessitates the exclusion of heart failure. And although the right side is more commonly affected, there is no consistency in the presentation to make the location of an effusion of benefit in identifying the etiology.

Despite the improvement in reaching the correct diagnosis by complete workup and management as suggested by the current guidelines, 6.5% of our study population remains undiagnosed<sup>20</sup>. However, exudative pleural effusion was more commonly encountered among our patients compared to transudative effusion. This finding is likely to be explained by the higher rates of PPE, malignant effusion, and TB as a cause of pleural effusion. Less common causes of pleural effusion were those secondary to hepatic hydrothorax, connective tissue diseases, and nephrotic syndrome. Enforcing the fact that pleural effusions are likely to be of exudative nature that require further workup to establish the underlying etiology.

In summary, we find the results of our study helpful in shedding the light on the changing clinical pattern of pleural effusion. Productive cough, dyspnea, and fever are the most common presenting clinical features but none of the symptoms were discriminatory in identifying an

etiology. Parapneumonic effusion was the most frequent cause of pleural effusion followed by CCF, malignancy, and TB. Patients with tuberculous pleural effusions were

significantly younger than the other groups. There was no significant difference in the duration of hospital stay among the groups.

## References

- Collins, TR, Sahn, SA Thoracentesis: Complications, patient experience, and diagnostic value. *Chest* 1987; 91:817.
- Storey DD, Dines DE, Coles DT. Pleural effusion: a diagnostic dilemma. *JAMA* 1976; 236:2183-86.
- Hirsch A, Ruffie P, Nebut M, et al. Pleural effusion: laboratory test in 300 cases. *Thorax* 1979; 34:106-12.
- N A Maskell, R J A Butland. BTS guidelines for the investigation of a unilateral pleural effusion in adults *Thorax* 2003; 58:ii8.
- Valdes L, Alvares D, Valle J, Pose A, San Jose E. The etiology of pleural effusions in an area with high incidence of Tuberculosis. *Chest* 1996; 109:158-62.
- Al-Qorain A, Larbi EB, Al-Muhanna F, Satti MB, Baloush A, Falha K. Pattern of pleural effusion in Eastern Province of Saudi Arabia: a prospective study. *East Afr Med J*. 1994; 71(4): 246-9.
- Al-Quorain A, Larbi EB, Satti MB, Al-Muhanna F, Baloush A. Tuberculous pleural effusion in eastern province of Saudi Arabia. *Trop Geogr Med*. 1994; 46(5): 298-301.
- Qari FA. Etiology of pleural effusion in Western Saudi Arabia. *Saudi Med J*. 2002; 23(3): 351-352.
- Light RW, MacGregor MI, Luchsinger PC, Ball WC. Pleural effusions: the diagnostic separation of transudates and exudates. *Ann. Intern Med* 1972; 77: 507-513.
- Light RW. Parapneumonic effusions and empyema. *Proc Am Thorac Soc*. 2006; 3(1):75-80.
- Tu CY, Hsu WH, Hsia TC, Chen HJ, Chiu KL, Hang LW, et al. The changing pathogens of complicated parapneumonic effusions or empyemas in a medical intensive care unit. *Intensive Care Med*. 2006; 32(4):570-6.
- Soriano T, Alegre J, Aleman C, Ruiz E, Vazquez A, Carrasco JL, et al. Factors influencing length of hospital stay in patients with bacterial pleural effusion. *Respiration*. 2005 Nov-Dec; 72(6):587-93.
- Sahn SA, Light RW. The sun should never set on a parapneumonic effusion. *Chest* 1989; 95(5): 945-7.
- Al Nozha MM, Al Maatouq MA, Al Mazrou YY et al. Diabetes mellitus in Saudi Arabia. *Saudi Med J*. 2004; 25(11): 1603-1610.
- Al Nozha MM, Al Khadra A, Arafah MR et al. Metabolic syndrome in Saudi Arabia. *Saudi Med J*. 2005; 26(12):1918-1925.
- Lian CK, Lim KH, Wong CM. Causes of pleural exudates in a region with high incidence tuberculosis. *Respirology* 2000; 5: 33-38.
- Alrajhi AA, Al-Barrak AM. Extrapulmonary tuberculosis, epidemiology and patterns in Saudi Arabia. *Saudi Med J*. 2002 May; 23(5):503-8.
- AlAmoudi OS. Prevalence of respiratory diseases in hospitalized patients in Saudi Arabia: a five year study 1996 - 2000. *Annals of Thoracic Medicine*. 2006; 1(2): 76-80.
- Medford A, Maskell N. Pleural effusion. *Postgrad Med J*. 2005 Nov; 81(961):702-10.
- Manuel Porcel J, Vives M, Esquerda A, Ruiz A. Usefulness of the British Thoracic Society and the American College of Chest Physicians guidelines in predicting pleural drainage of non-purulent parapneumonic effusions. *Respir Med*. 2005 Oct 24; 81(14): 112-18.

# Clinical Profile of HIV/AIDS Patients in Jammu

Sourabh Verma, Annil Mahajan, JB. Singh, Manoj Sharma.

## Abstract

The present prospective study was conducted for the first time to evaluate the clinical profile of HIV/AIDS patients in this region over a period of one year. Cases were diagnosed as per the NACO, 2000 criteria. Among 177 patients enrolled; 72.8% were males and 27.1% females with male: female ratio was 2.68:1. Highest incidence of HIV/AIDS was found in the age group of 31–40 years (48.58%) followed by 21 – 30 years (30.50%). Paramilitary personnel formed the major group (31.7%) followed by housewives (27.64%), drivers (17.05%) and labourers formed 12.94% of the patient population. 132 (74.57%) of the patients were married with live partners and 22 (12.42%) were married but widowed. 23 patients (12.99%) were unmarried out of which 7 were children below age of 10 years. All the females affected had HIV positive living/expired partner. 89.26% of cases were heterosexual, no homosexual, but in 6.21% of the patients mode of transmission was unknown. Vertical transmission was responsible in 3.95% of the cases. Blood transfusion was responsible only in 0.56% (1 patient). The maximum number of patients in our study group presented with fever 48.58% which was followed by diarrhea 27.68%, weight loss 21.46%, while 22.03% of patients were asymptomatic. 27 patients had tuberculosis at some stage of HIV/AIDS infection out of which 11.3% had active disease and were on ATT. 7 patients (3.9%) had already been treated with ATT after being diagnosed with HIV infection. Generalized weakness, cutaneous manifestations, lymphadenopathy, behavioural abnormalities & bleeding per rectum was seen in 10, 9, 8, 7 & 5 patients respectively and small number of patients had mucosal lesions (candidiasis-4), arthralgias (4); pain abdomen (3), pelvic inflammatory disease (3), loss of appetite and orchitis one each along with the chief presentation. The numbers of patients having total lymphocyte counts less than 1200 were 60 (33.89%); while numbers of patients with CD4 count less than 200 were 78. HBsAg was positive in 6 cases (3.38%); anti Hepatitis C antibody was present in none of our cases while no patient in our study presented with hepato-biliary disease. 7 (3.95%) out of 177 cases were positive for VDRL. HAART was given to 98 patients (55.36%) along with prophylaxis for opportunistic infections and 53 cases (29.94%) were given prophylaxis for opportunistic infections only. Rest of the patients did not require any medication. The present study highlights the increasing burden of HIV/AIDS patients contrary to under reporting in the past in this region. The results may be useful in developing various programs for health promotion in patients of HIV/AIDS from this region.

JK-Practitioner 2006;14(2):79-83

## INTRODUCTION

The Acquired Immunodeficiency Syndrome (AIDS) caused by a virus known as Human Immunodeficiency Virus (HIV) is a disorder of immune deregulation that leads to a continuous spectrum of manifestations, ranging from an overactive and misdirected immune response to severe cellular immunodeficiency. The first case was detected in USA in 1981 and HIV / AIDS epidemic continues its expansion across the globe with approximately 14,000 new HIV infections a day. Thus burden of HIV/AIDS is continuously increasing and it is predicted that incidence of HIV infection will increase with at least 45 million new infections between 2002 and 2010. The first case of HIV infection in India was detected in 1986. It is estimated that number of people living with HIV in India could be around 5.1 million.

Numerous studies from India and outside India have suggested varied clinical profile and associated infections like tuberculosis, hepatitis B, hepatitis C and syphilis in HIV/AIDS patients. Hence, there is a need to define disease aspects such as spread, clinical manifestations, natural course so that management strategies could be planned early for facilitating better primary and preventive care and timely introduction of opportunistic infection prophylaxis to such patients. Although extensive studies have been done in west; there is a dearth of such Indian data particularly from this region. Moreover, according to HIV prevalence rates in adult population, Jammu & Kashmir has been placed in low prevalence state. However, in J&K also disease has begun to show its ugly face in the recent time. Hence, the present one year prospective study was planned to evaluate clinical profile of HIV/AIDS in Jammu and to find out its association with associated infections like tuberculosis, hepatitis B, hepatitis C and syphilis.

Key words: HIV, AIDS, HAART

## Authors affiliations:

Sourabh Verma, Annil Mahajan, JB. Singh, Manoj Sharma.

Postgraduate Department of General Medicine

Govt. Medical College Jammu

India- 180001

## Accepted for Publication

November 2006

## Address for correspondence

Dr. Sourabh Verma

Postgraduate Student

Department of Medicine

Govt. Medical College Jammu-180001

E.mail:sourabh\_verma2000@yahoo.com.

**MATERIAL AND METHODS**

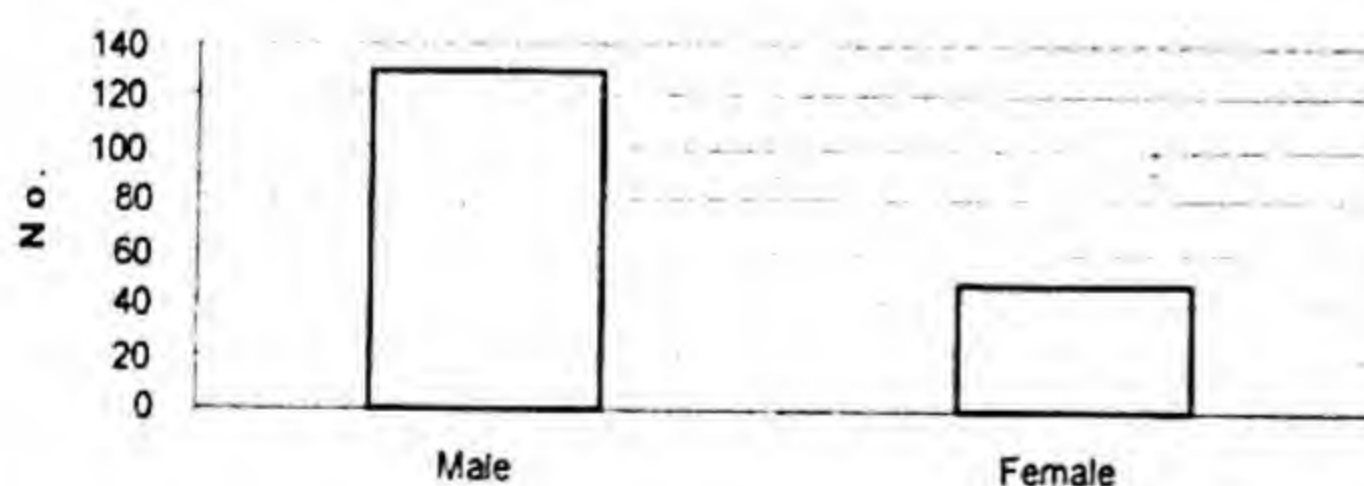
The present study was conducted in the Government Medical College, Jammu from April, 2004 to March, 2005. The protocol according to the WHO/NACO guidelines was followed in the present study. All the subjects who attended VCTC during the above mentioned period were considered for participation. After doing pretest counseling, written consent was taken from adults. In case of children, parental consent was taken for getting enrolled in the study. The subjects under study either presented themselves or were directed to the VCTC from OPD/indoor/wards in Govt. Medical College and associated hospitals. The detailed clinical history, physical examination and laboratory investigations for all patients were recorded on a special pre-designed performa. All patients were subjected to number of laboratory tests routinely according to the WHO/GOI guidelines (Strategy II and III as per NACO).

Serum was subjected once to Rapid Test and ELISA and if negative, serum was considered free of HIV and if positive was taken as HIV infected for all practical purposes. The case was defined as an AIDS case as per (NACO, 2000) criteria. The following screening tests were used to evaluate associated infections, HbsAg micro screen ELISA Test Kit (for hepatitis B); HCV micro ELISA (3rd generation): for detection of antibodies to Hepatitis C virus in human serum / plasma and Rapid plasma reagin test (RPR Test): for rapid serological diagnosis of syphilis. Tuberculosis was diagnosed as per standard protocol.

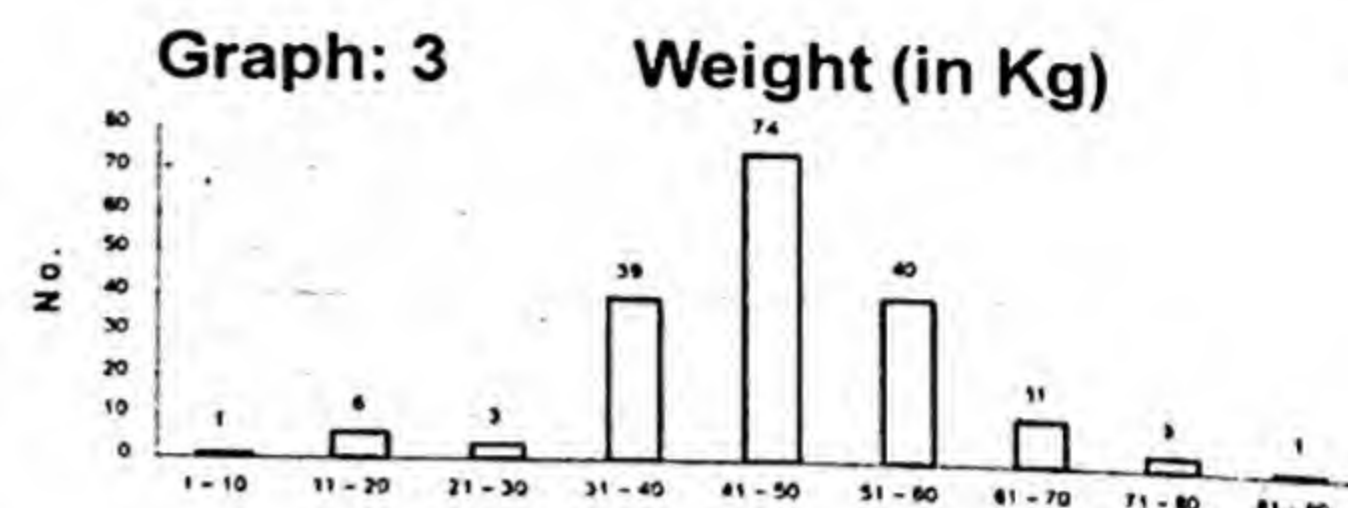
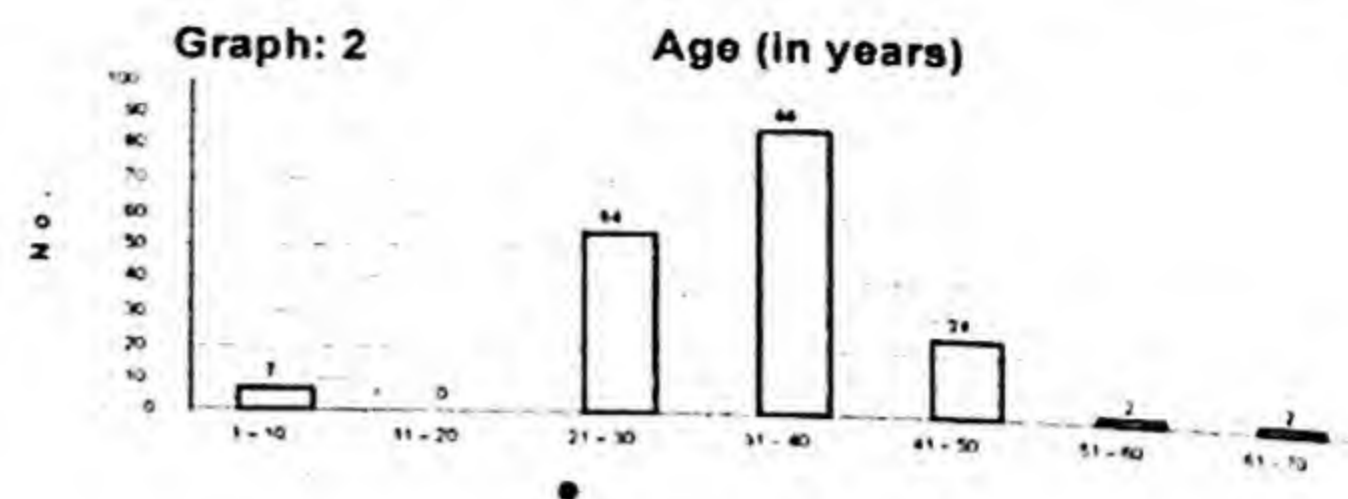
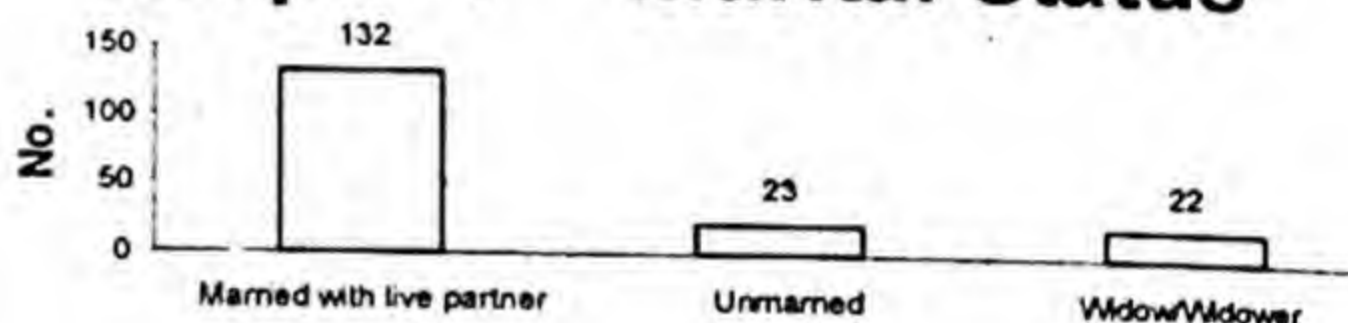
Patients were followed for at least 3 months after date of enrollment in the study. Total/Absolute lymphocyte count (TLC/ALC) was done in all the cases and CD<sub>4</sub> count in selective cases, wherever it was feasible. Patients with less than 1200 total lymphocytes/mm<sup>3</sup> and/or CD<sub>4</sub> count < 200 were put on anti-retroviral drugs and responses were followed.

**RESULTS**

The present study enrolled 177 HIV/AIDS patients. Out of these patients; 72.88% were males; while 27.11% were females (Fig-1). Majority of the patients were in the age group of 21-40 years (Fig-

**Graph 1: Sex**

2). Maximum (74) cases were in 41-50 kg weight category (Fig-3). 74% of the patients were married with live partners while 12.99% were unmarried. 12.42% patients were widows who had lost their husbands because of the disease 6 months-2 years prior to the study period (Fig-4). As far as the profession is concerned paramilitary personnel (including Police, CRPF and BSF) formed the

**Graph: 4 Marital Status**

major group which accounted for the 31.7% cases. 27.64% of the infected individuals were housewives while drivers formed 17.05% and laborers formed 12.94% of the patient population (Table-1). Heterosexual mode of transmission was the commonest mode in our study with 89.26% falling in this category. Blood transfusion formed only 0.56% as the causative source while no intravenous drug abusers or homosexuals was found in the present study. Vertical transmission (mother to child) was responsible for 3.95% of

**TABLE 1: PROFESSION**

Profession	No.	%Age
Paramilitary (Police, CRPF, BSF)	53	31.17
Labourer	22	12.94
Agriculturist	2	1.17
Businessman/Private Jobs	10	5.88
House wife	47	27.64
Driver	29	17.05
Army	4	2.35
Religious Preacher	3	1.76
Total	170*	100

\* Excludes 7 children

cases while in 6.21% of our cases; mode of exposure remained unknown (Table-2).

In the clinical presentation, it was found that fever was present in highest number of our patients i.e. 48.58%; while diarrhoea was present in 27.68% of cases and loss of weight in 21.46% of cases. 22.03% of our cases were asymptomatic. Associated tuberculosis was found in 15.25%

**TABLE 2: MODE OF EXPOSURE**

Mode of exposure	No.	%age
Heterosexual	158	89.26
Homosexual	0	0.00
Blood transfusion	1	0.56
Vertical	7	3.95
Unknown	10	6.21
Total	177	100

of cases while lymphadenopathy was present in 4.52% of cases. Cutaneous manifestations were associated with 5.08% cases and general weakness was associated in 5.64% of cases. Other manifestations like behavioural abnormalities, pain abdomen, mucosal lesions, loss of appetite, orchitis, bleeding per rectum, arthralgia, and pelvic inflammatory disease were present in 15.8% of cases (Table-3).

We did total lymphocytic count in all the cases and Cd4 cell counts in 129 cases (72.88%). Number of patients having total lymphocyte counts less than 1200 were

**TABLE 3: PRESENTATION**

Presentation*	No.	% age
Fever	86	48.58
Diarrhoea	49	27.68
Loss of weight	38	21.46
Asymptomatic	39	22.03
Tuberculosis <sup>b</sup>	27	15.25
General Weakness	10	5.64
Cutaneous Manifestations	9	5.08
Lymphadenopathy	8	4.51

**Others**

● Muscosal lesions (condidiasis)	4	2.25
● PID (Pelvic Inflammatory Diseases)	3	1.69
● Arthralgia	4	2.25
● Bleeding per Rectum	5	2.82
● Loss of Appetite	1	0.56
● Behavioural Abnormalities	7	3.95
● Pain Abdomen	3	1.69
● Orchitis	1	0.56

\* More than one presenting features present in patients.

<sup>b</sup> Active pulmonary tuberculosis was present in 17 cases, 3 had active extrapulmonary tuberculosis, 7 cases had suffered from tuberculosis and had finished their treatment before being enrolled in the study.

60(33.89%); while number of patients with CD4 count less than 200 were 78; out of the 129 cases in which CD4 counts were done which comes out to be 60.46%. In the present study, we did not find any significant correlation between CD4 count and absolute lymphocyte count (Fig-5, Table-4).

**TABLE 4: ABSOLUTE/TOTAL LYMPHOCYTE COUNT**

Absolute lymphocyte count	No.	%age
<1200	60	33.89
>1200	117	66.10
Total	177	100

Co infection with conditions which have similar mode of transmission as HIV like hepatitis B, hepatitis C and syphilis

formed 7.33% of the cases; out of which 3.38% were positive for hepatitis B surface antigen (HbsAg) and in 3.95% of cases, VDRL was positive.

**Graph: 5 CD4 Counts**

No case in our study had coinfection with hepatitis C virus (Table-5). In our study, out of the 177

**TABLE 5: ASSOCIATED INFECTION WITH HEPATITIS B, HEPATITIS C AND SYPHILIS**

	+ve	-ve
HbsAg	6	171
VDRL	7	170
Anti HCV	0	177

cases, highly active antiretroviral therapy (HAART) was given to 98 patients (55.36%) along with prophylaxis for opportunistic infections and 53 cases (29.94%) were given prophylaxis for opportunistic infections only. Rest of the patients did not require any medication (Table-6).

**TABLE 6: MEDICAL MANAGEMENT RECEIVED**

	No.
HAART* (Highly Active Anti – Retroviral Therapy)	98
Prophylaxis for opportunistic infections	53

\* All patients on HAART were given prophylaxis for opportunistic infections also.

**DISCUSSION**

The male: female ratio in the present study was 2.68:1. The study done by Rothenberg *et al* (1987) reported a very high predominance of involvement of males i.e. 90.5% in their study in contrast to females (9.5%). In a large Indian studies Ghate *et al* (2002) reported M: F ratio of 2.22:1. Whereas, Kumarasamy *et al* (2003) reported M: F ratio to be – 2.6: 1 which is consistent with our study. It seems that more and more females are contracting the disease with the passage of time. Kumarasamy *et al* (1995) found that 85% and Ghate *et al* (2002) reported 88.1% of patients in the age group of 21 – 40 years. The incidence was little lower in the present study than these studies (Fig-2). This can be explained on the fact that both these studies were conducted in metro cities unlike present study from small region. The other point to explain this difference can be the diverse socio-economic, conditions, moral values and sexual behavior prevailing in different communities.

In our study, in male patients, 29 patients (17.05%) were vehicle drivers and labourer class (12.94%). Kothari *et al* (2001) reported the occupation of involved patients as transport – 40%, labourers – 20% and Migration – 43%, which is not in tune with our findings; because in Jammu, though the migration is great from states like Orissa, Bihar, Uttar Pradesh, but most of them move in with their families and observe traditional social and moral codes. We in our study did not have a single patient who was from migration group. The difference in transport and labourer group is also very glaring this may be explained on basis of better awareness among these people which is an encouraging sign.

The present study was conducted in J&K which being a disturbed area, large deployment of Police, Central Reserve Police Force (CRPF), Border Security Force (BSF) and Army is present who have served in different parts of the country and moreover; most of patients from military and paramilitary services are already diagnosed and referred to us for assessment and management. So, the highest number 53 patients (31.17%) in these people can't be statistically included in the clause of profession of our state. However their age, sex, clinical profile have all been included in the study. Hira *et al* (2003)<sup>11</sup> found that the skilled and unskilled labourer group formed a major chunk of people infected (56.3%) which is consistent with our estimate of infected population. The difference in percentages can be explained by local factors as explained earlier.

In the present study, marital status (Fig-4) is nearly coinciding with the findings of Hira *et al* (2003)<sup>11</sup> and Mukhopadhyay *et al* (2001)<sup>12</sup>. In order to assess vertical transmission from mother to child; we screened all the children of HIV positive females between age of 1–10 years born to HIV positive females and found that seven of them were also positive (3.95%).

In the present study the data regarding the mode of exposure (Table-2) varied to some extent from previous reports. Hira *et al* (2003)<sup>11</sup> in their study reported that 88.7% of their patients were having heterosexual mode of transmission while homosexual accounted for 0.9%, bisexual accounted for 9% of patients. In another Indian study by Kumarasamy *et al* (2003)<sup>13</sup> heterosexual mode of transmission in 95% was responsible, blood transfusion accounted for 4.6% of cases; while infected needles/syringes were responsible for transmission in 2.4% of cases. Our data is fairly consistent with these reports as far as the predominant mode of transmission i.e. heterosexual transmission is concerned.

Hira *et al* (2003)<sup>11</sup> reported that in 9% of their cases mode of transmission was unknown. Kumarasamy *et al* (2003)<sup>13</sup> were able to record mode of transmission in every patient. The disparity can be due to difficulty to get the proper information regarding socially unacceptable sexual practices indulged by different individuals. Moreover there might be other modes of transmission about which neither the patient nor the doctor are certain. Even in one of the recent European study by Sabin *et al* (2004)<sup>14</sup>, the reported incidence of unknown mode of transmission was found to be in range of 4.3% to 9.1%. Blood transfusion was responsible in 12.16% of cases reported by Sircar *et al* (1998)<sup>15</sup>, while NACO in their publication report blood transfusion as mode of transmission in 8.1% of the cases unlike present study recording very low percentage. This can be explained by the fact the in J&K state, most of the transfusion is done in the Government institutions, where it has been made mandatory to test for HIV quite early since 1991. Sircar *et al* (1998)<sup>15</sup> reported homosexuality as mode of transmission in 1.2% and national average reported by NACO is 0.5% which is almost consistent with our average of 0.56%. Msellati *et al* (1995)<sup>16</sup> in a multinational study involving Europe, Africa and America reported the incidence of mother to child transmission as 25.45% in developing countries but in our study the incidence was 3.95% only. This difference can be

explained by the fact that after 1995 even developing countries are testing pregnant females in secondary and tertiary care hospitals much more frequently and adopting the prophylactic protocols.

As far as the clinical presentation of our patients goes (Table-3) the first thing which is very pertinent is that all the patients had more than one presenting feature variation were recorded from the previous reports. In our study, the highest number of patients had fever as one of the presenting feature i.e. 48.58% which is consistent with the previous studies<sup>11,12,13,14</sup> although there is some difference in percentage of patients. Reeve *et al* (1989)<sup>17</sup> have reported fever in 49.4% of their cases which is very near to our figures; though weight loss was the chief complaint (i.e. in 78.6% of the cases) in their study. In our study loss of weight was present in 21.46% of cases.

In our study 22.03% (39 patients) were totally asymptomatic; while Ghate *et al* (2002)<sup>18</sup> found the incidence of patients who were reportedly asymptomatic as 45.98% (1997), 46.6% (1998) and 67.6% (1999). This difference is very big but it can be explained by the fact that the study was conducted in HIV Reference Clinic of the National AIDS Research Institute, Pune which is a referral centre. It is quite understandable that asymptomatic phase in HIV infected patients is very long and there must be much higher number of asymptomatic patients in our population also. As the general screening of the population cannot be done without counseling and written consent of the patient, it needs good public awareness and education so that more and more people come to voluntary counseling and testing centers. The incidence of diarrhoea reported in different studies varies from 7.5% to 37.0% of cases<sup>11,13,14,15</sup>. This variation can be explained on the basis of the difference in hygienic status of different populations and the local quality of drinking water supply. May be the cotrimoxazole prophylaxis also contributes to the decline in number of patients presenting with various diarrhoeal illnesses associated with HIV infection. The incidence of weight loss reported in different studies is as follows: Sircar *et al* (1998)<sup>15</sup> -74.1% cases, Ghate *et al* (2002)<sup>18</sup> -2.1% (1997); 1.44% (1998), 3.6% (1999) of the cases, Reeve *et al* (1989)<sup>17</sup> -78.6% of the cases. Our group of patients 21.46% cases had weight loss which is close to study by Hira *et al* (2003)<sup>11</sup> who reported the incidence to be 23.5%.

In other associated features, tuberculosis formed the major group with 15.25% (27 patients) in present study. In other Indian studies; Sircar *et al* (1998)<sup>15</sup> reported tuberculosis in 54.8% cases; Ghate *et al* (2002)<sup>18</sup> in 7.4%-11.8% of the cases and Hira *et al* (2003)<sup>11</sup> reported tuberculosis in 25.4% of the cases. The highest incidence reported by Sircar *et al* (1998)<sup>15</sup> seem to be due to the fact that out of the 74 HIV seropositive patients selected for the study, 83.7% (62 patients) had AIDS. Moreover; as concluded by Ghate *et al* (2002)<sup>18</sup>, clinical conditions like oral thrush, tuberculosis, skin rash and sexually transmitted diseases (STD) are showing decreasing trends over the years. The lower incidence of tuberculosis in our study as compared with Hira *et al* (2003)<sup>11</sup> can be attributed to the early diagnosis, increasing awareness and high index of

suspicion about the presence of primary disease and opportunistic infections and timely prophylaxis.

As per the guidelines issued by the World Health Organisation (WHO) April 2002<sup>17</sup> for starting antiretroviral therapy in resource limited settings; it is recommended that total lymphocyte count (TLC) of less than 1200 may be used as a substitute indication of starting treatment. We also did total lymphocyte count in all the cases (Graph-5; Table-4). Though absolute lymphocyte count may be a fair substitute for starting treatment in resource limited settings, in our study we did not find that absolute lymphocyte counts of less than 1200 and CD4 count less than or equal to 200 correlates well as per given above figures.

Kothari *et al* (2001)<sup>9</sup> in their study found no case who was HbsAg positive. Kumarasamy *et al* (2003)<sup>11</sup> in their study found that 6% of patients were coinfecting with Hepatitis B virus (HBV) and 4.8% were coinfecting with Hepatitis C virus (HCV). Sud *et al* (2001)<sup>20</sup> in their study found that 33.8% of cases were infected with Hepatitis B virus. As is clear from the above mentioned Indian studies, incidence of hepatitis B and hepatitis C virus coinfection varies from 0% to 33.8% from study to study conducted at various places from time to time. Probably this difference may be due to the varied prevalence of hepatitis B and C

virus infection in different populations as such. As the route of transmission of hepatitis B and hepatitis C viral infection is virtually same as the route of HIV infection; the low incidence in our study can be explained on the basis of the fact that no case in our study was intravenous drug abuser and only one case (0.56%) of HIV infection had blood transfusion as route of infection. Kothari *et al* (2001)<sup>9</sup> in their study reported co infection with syphilis in HIV positive cases in 3.3% of cases while in the study by Deivanayagam *et al* (2001)<sup>21</sup> reported the incidence of syphilis to be 9.31%. In our study; we found the coinfection with syphilis in 3.95% of cases which is near to the incidence by Kothari *et al* (2001)<sup>9</sup>.

### CONCLUSION

Present study highlights the increasing burden of HIV/AIDS patients contrary to under reporting in the past in this region. Highest incidence reported in males with age group of 31 to 40 yrs. Heterosexual mode of infection was the most common. Paramilitary personal followed by house wives and drivers were the commonest group affected. Fever was the most common complaint. TB was associated in 15.25% of the cases. Results of the present study in the areas like J&K are an eye opener for health promotion providers and stress the need to formulate strategies & policies to combat the menace of this dreadful disease.

### References

- Orenstein R. Presenting syndromes of human immunodeficiency virus. *Mayo Clin Proc* 2002; 77 : 1097-1102
- National AIDS Control Organisation, Ministry of Health & Family Welfare, Govt. of India, New Delhi. National History and Clinical manifestations of HIV/AIDS. Specialist training and reference module 2000 pp: 104 - 110.
- UNAIDS. Regional HIV / AIDS statistics and features end of 2003. *AIDS Epidemic Update* 2003; pp. 1-12.
- Stover J, Walker N, Garnett GP *et al*. Can we reverse the HIV / AIDS pandemic with an expanded response? *Lancet* 2002; 360 : 73-77
- Simoes EAF, Babu GP, John TJ *et al*. Evidence for HTLV-3 infection in prostitutes in Tamil Nadu (India). *Ind J Med Res* 1987; 85 : 335-338.
- National AIDS Control Organisation. Combating HIV / AIDS in India 2003-2004; Ministry of Health and Family Welfare.
- Rewari BB, Joshi PL. Epidemiology of HIV / AIDS - reference to India. In: Das S (ed.) *Medicine Update* Vol.13. Association of Physicians of India. Mumbai 2003 pp: 79-82.
- National AIDS Control Organisation, Ministry of Health & Family Welfare, Govt. of India, New Delhi. Clinical case definition for AIDS. Specialist training and reference module 2000 pp: 33.
- et al*. Survival with the acquired immunodeficiency ZZZ syndrome - experience with 5833 cases in New-York city. *N Eng J Med* 1987; 317:1297-1302
- Ghate MV, Divekar AD, Risbud AR, Thakar MR, Brahme RG, Mehendale SM. Changing trends in clinical presentations in referred human immunodeficiency virus infected persons in Pune, India. *JAPI* 2002; 50 : 671-673.
- Kumarasamy N, Solomon S, Flanagan TP, Hemalatha R, Thyagarajan SP, Mayer KH. Natural history of human immunodeficiency virus disease in Southern India. *Clin Infect Dis* 2003; 36 : 79-85
- Kothari K, Goyal S. Clinical profile of AIDS. *JAPI* 2001; 49 : 435-438.
- Hira SK, Shroff HJ, Lanjewar DN, Dholkia YN, Bhatia VP, Dupont HL. The natural history of human immunodeficiency virus infection among adults in Mumbai. *Nat Med J Ind* 2003; 16 (3) : 126-131.
- Mukhopadhyay C, Nath G, Gulati AK, Mohapatra SC. Prevalence of HIV among low and high risk population of eastern part of northern India. *J Commun Dis* 2001; 33 (2) : 136-142.
- Sabin CA, Smith CJ, Gumley H, Murphy G, Lampe FC, Phillips AN, Prinz B, Youle M, Johnson MA. Late presenters in the era of highly active antiretroviral therapy : Uptake of and responses to antiretroviral therapy. *AIDS* 2004; 18:2145-2151.
- Sircar AR, Tripathi AK, Choudhary SK, Misra R. Clinical profile of AIDS : A study at a referral hospital. *JAPI* 1998; 46 (9) : 775-778.
- Msellati P, Newell ML, Dabis F. Rates of mother to child transmission of HIV-1 in Africa, America and Europe : Results from 13 perinatal studies. *J Acquir Immune Def Syndr* 1995; 8 : 506-510.
- Reeve PA. HIV infection in patients admitted to a general hospital in Malawii. *Br Med J* 1989; 298 : 1567-1568
- WHO. Department of HIV / AIDS. Scaling up anti-retro viral therapy in resource limited settings. Guidelines for a public health approach. June 2002; (V), pp. 25.
- Sud A, Singh J, Dhiman RK, Wanchu A, Singh S, Chawla Y. Hepatitis B virus co-infection in HIV infected patients. *Trop Gastroenterol* 2001; 22 (2) : 90-92.
- Deivanayagam CN, Rajesekaran S, Senthilnathan V, Krishnaraja Sekhar OR, Raja K, Chandrasekar C, Palanisamy S, Dinesh AS, Jothivel G, Elango SV. Clinico-radiological spectrum of tuberculosis among HIV seropositives - A tambaram study. *Ind Tubercul* 2001; 48 : 123-127

# Complication of Variable Screw Placement System (VSP) In Instrumented Fusion Of Non-Traumatic Lumbosacral Spinal Disorders

Butt M F, Hakeem I.H, Halwal M.A, Munir Farooq, Mir M. R. , Kanngu Khursheed A.

## Abstract

We analyzed the complications encountered in fifty consecutive cases who had undergone posterolateral lumbosacral fusion with pedicle screw fixation for non traumatic disorders after a minimum follow up of two years. Of these forty eight had Spondylolisthesis (isthmie and degenerative) and other two had lumbar disc disease with instability. There were 6 multilevel and 44 single level fusions.

There were forty-eight individual complications including pseudoarthrosis. The complications which were encountered in our study during the operative and the postoperative follow up were screw misplacement, wound infection, pseudoarthrosis, neural injury, loosening of the implant, bending and the breakage of the screws.

JK-Practitioner 2007;14(2):84-87

## INTRODUCTION

Ever since Boucher<sup>1</sup> described pedicle screw fixation for spine, internal fixation devices for spinal disorders are in a phase of evolution. All spinal systems to date have experienced significant problems and complications, and an ideal system is yet to develop search is on for an ideal system. An ideal system would allow for rapid patient mobilization and comfort, and minimum of pseudoarthrosis, wound infection, iatrogenic complications and operative time<sup>2</sup>.

Instrumented fusion for non-traumatic disorders of the spine has gained wide acceptance<sup>3,4,5</sup>. Pedicle screw fixation systems are superior to earlier devices in that less motion segments are involved in a shorter fusion construct and three column fixation can more powerfully control coronal, sagittal and rotational deformities. Potential disadvantages include complications related to instrumentation.

There are numerous studies<sup>6-12</sup> on the complications associated with the use of pedicle screw instrumentation. The incidence of mechanical failure due either to loosening or implant fatigue is between 5 to 31% in various studies followed for variable periods of time<sup>13-15</sup>.

This article presents complications observed in 50 cases of instrumented fusion of non-traumatic disorders of spine over a period of three years

## PATIENTS AND METHODS

Between Jan 2001 and Sept 2003 fifty consecutive patients were treated by posterolateral fusion and Steffee transpedicular instrumentation for non-traumatic spinal disorders. Out of these 34 were females and 16 males. The mean age at the time of the surgery was 38 years. The main indication for the surgery is depicted in table I.

Table I	
Condition	No of cases (%)
Low grade isthmie spondylolisthesis	35(70)
High grade isthmie spondylolisthesis	08(16)
Degenerative spondylolisthesis	05(10)
Lumbar disc disease and instability	02(04)

Preoperative assessment involved clinical, functional and radiographic evaluation. Pain and functional grade for VSP studies was used. MRI scans were used to detect herniated discs at the intended level of the fusion or the level above, or detect the spinal canal as also to exclude other causes of back pain and sciatica.

The patients were put prone on the bolsters on an operating table.

**Key Words:** -spondylolithesis, pedicle screw fixation, posterolateral fusion, pseudoarthrosis, VSP fixation Systems.

## Authors' affiliations:

Mohammad Farooq Butt, Hakeem I.H, M.A. Halwal, Munir Farooq, Prof. Mohammad Ramzan Mir, Khursheed A Kangu

Government Hospital for Bone & Joint Surgery Srinagar 190005

## Accepted for Publication :

December 2006

## Correspondence:

Mohammad Farooq Butt  
Cell No. 9419004007

Email mfbutt72@yahoo.co.in

Fax No.0194243730

Standard midline incision and subperiosteal dissection was used for posterior exposure. Anatomical landmarks according to Roy Camille's method<sup>11</sup> were used to identify the entry point of the pedicle screw. The pedicle entry point was made with the help of an awl and its position confirmed on the C arm image in the lateral plane. The awl was also used for probing the intactness the pedicle walls. Subsequent steps of tapping the posterior part of the pedicle, insertion of 5.5 mm diameter 38-40 mm length screw and placement of proper size plate after adequate contouring followed.

All the patients had the posterolateral fusion using the autologous iliac graft from the posterior iliac crest. No attempt for the reduction of the slip was made in the cases with low grade spondylolisthesis, and the fusion was by and large attempted in situ in these cases. Parenteral antibiotics were used for three days and patients mobilized on a lumbar corset. Patients were followed regularly and clinical, functional and radiological assessment was carried out.

The implant removal after 18 months of the surgery was not made routine and was carried out only in cases presenting with complications arising after the fixation like loosening and breaking of the screws and pain because of the hardware.

All the patients were followed clinically and radiologically for at least two years after the primary procedure, patients with less than two years of follow-up were not included in this study. The follow up averaged 32 months

At the final follow-up pain and functional grades were recorded and compared with the preoperative status. The clinical results were evaluated by the criteria of Kim and Kim<sup>12</sup>. The criteria for the fusion were continuous bony bridging verified at all intended sites, and anatomic grade of Massachusetts General Hospital Anatomic, Economic, and Functional (AEF) rating system was used for fusion assessment (as has been adopted by Dawson EG et al<sup>13</sup>).

## RESULTS

The average pain score at the final follow-up was 3.7 [range 3-5] whereas the preoperative pain score was 2.4 [range 1-3]. The improvement in functional score was from a preoperative average of 2.7 [range 2-4] to 4.2 [range 3-5]. As per the anatomic grade of AEF rating system [vide supra] only two patients achieved A4 fusion. Fourteen patients had A3 and twenty six patients achieved A2 fusion. Eight patients with fusion status of A1 were labeled as fusion failures. As per Kim and Kim criteria good to excellent results were seen in 70% of cases.

Pseudoarthrosis being an outcome not directly related to device was taken a separate entity. Therefore there were thirty implant related complications in twenty-three patients. Pseudoarthrosis was seen in 15 cases. Neurological injury attributed to pedicle screw fixation was seen in 4 patients, and wound infection was seen in 4 cases. Implant related complications are depicted in Table II and are discussed in detail.

Table II

Complications	No of Complications	No of patients with complications
Screw malplacement	04 [08%]	03[06%]
Screw bending	05 [10%]	03[06%]
Screw breakage	08 [16%]	04[08%]
Screw loosening	02 [04%]	02[04%]
Pseudoarthrosis	15 [30%]	15[30%]
Superficial wound infection	05 [10%]	05[10%]
Deep wound infection	02 [04%]	02[04%]
Neurological injury	04 [08%]	04[08%]
Graft site pain	03 [06%]	03[06%]

**SCREW MALPLACEMENT:** This was observed in 3 patients. In one of the patients both L5 screws violated the medial pedicle walls were confirmed by post fixation CT scan. One L5 screw was placed into the disc space and had only partial purchase in the pedicle. Another S1 screw was likewise misplaced into the disc space.

**SCREW BENDING:** Five screw bending were seen in three patients. In two patients, both involving multilevel fusion, bending in both sacral screws was noted. This probably occurred during the final seating of plate over the screws. Similarly an isolated right L5 screw bending was seen in another patient.

**SCREW BREAKAGE:** Eight pedicle screws broke in four patients. In one case both lumbar screws broke at the isthmus. Ipsilateral lumbar and sacral screw were fractured in another case. One case each of isolated lumbar and sacral screw breakage was seen. A case of high grade slip reduction and fusion presented with breakage of both sacral screws. No plate fractures were reported.

**SCREW LOOSENING:** A screw was said to be loose when a continuous lucency was seen at the bone screw interface 1mm or more wide and surrounded by a thin sclerotic zone. We observed two screw loosening in sacral pedicles, both resulting in backing out presenting as painful subcutaneous hardware.

**PSEUDOARTHROSIS:** Radiologically acceptable fusion was attained in 72% of cases and rest of them were labeled as fusion failures.

**NEURAL COMPLICATIONS:** A transient S1 neuropraxia was seen in two patients which resolved in the postoperative period. In one case of grade IV listhesis weakness of L was seen which did not recover, and was attributed to reduction of the slip. An EHL weakening of two MRC grade was seen in another patient in the immediate postoperative period, but no significant recovery was recorded afterwards.

**WOUND INFECTION:** Two cases of deep wound infection were seen, growing *Pseudomonas aeruginosa* and *Staphylococcus aureus* on laboratory cultures. Fortunately they responded to appropriate antibiotics. Superficial wound infection in five patients resolved with antiseptic dressings.

**GRAFT SITE PAIN:** Three patients complained of this problem. Only in one case analgesics were being used for pain relief whereas in other two symptoms resolved within three months of surgery.

## DISCUSSION

Pedicle screws combined with plates, rods or external fixators have become a prevalent form of spinal fixation system in the world<sup>14</sup>. These systems allow segmental fixation of the spine for several conditions such

as spondylolisthesis, tumors, instability after trauma, multilevel laminectomy and degenerative arthritis. This technique is especially useful when posterior elements are unsuitable for fixation by hooks or sub laminar wires, and it may permit fixation over shorter spinal segments compared with traditional hook-and-rod type system. The use of pedicle screw system, however, is not without complications. Concerns over surgical complications, safety and efficacy have surfaced. Fatigue failure of implant, infection, neurological damage is amongst the major complications reported by a number of researchers.

Our screw misplacement rate of 8% is probably less because we did not routinely use CT scan to confirm the position of the pedicle screws. A screw misplacement rate of 10-28.8% has been reported by researchers<sup>1</sup>. Overall there were 33 instrument related complications in 26 patients and fifteen fusion failures. This amounts to a high rate of failures. Soini J et al<sup>2</sup> reported a complication rate of 57% in 51 patients who had spinal fusion using the AO device in disorders such as lytic or degenerative spondylolisthesis and spinal stenosis. Screw breakage occurred in four patients. Fatigue failure indicates the micro motion at the region of the screws and the plates, wrong angle of placement, improper angle between the screw and the plate and inadequate plate contouring. The relation between the fatigue failure and the non union is controversial. Pihlajamaki et al<sup>3</sup> reported a high rate of screw breakage in multilevel fusions than in single level fusion. Multiple level fusions without the screws in the intermediate segments are more susceptible to the screw loosening. We observed four screw failures in two patients with multilevel fusion. But a significant no of complications were seen in single level fusions as well.

Accurate determination of fusion status has been an unsolved problem in spinal surgery<sup>4</sup> and it can not be described entirely a device related complication. In the assessment of fusion in VSP studies, the overlying

shadow of the plates adds to the uncertainty. Because of these reasons for failure to label pseudoarthrosis and the numerous reasons for non-correlation of fusion and clinical success, the primary evaluation of surgical results, as observed by some, should continue to be based on clinical grounds<sup>5</sup>. Our non-union rate of 28% is higher than many studies of instrumented fusion for non-traumatic disorders of the spine. Steffee et al<sup>6</sup> in their study reported a union rate of 91.6%, whereas non-union rate in another study<sup>7</sup> was 18%. Our poor results with fusion could be explained by the learning curve and that some of the surgeries were done by the residents.

In contrast to a previous study<sup>8</sup>, infection was not a major concern in our study and in no case exploration, incision drainage or removal of implant was needed to treat the infection. The low infection rate in our study could be because we operated young patients, and there were no case of tumors or metastatic disease. We also did not encounter any case of pedicle fracture, vascular injury or plate failure.

Neurological related complication is a major concern with transpedicular fixation devices, particularly when reduction of slipped vertebrae is attempted. We attributed neurological problem in one patient to the reduction of slip.

The chance of the metal related complications with a little evidence for the augmentation of the fusion by the implant are still the main drawbacks of the procedure and a lot needs to be done to improve the procedures related to the fusion in the non traumatic spinal disorders and the implants used in these procedures. The high rate of complications seen in transpedicular instrumentation suggests that it is a technically demanding procedure, and lack of experience adds to the rate of complications. If technical inadequacies are addressed the no of complications can be reduced, fusion rate may rise, resulting in overall clinical and functional improvement.

## References

1. Boucher HH. A method of spinal fusion. *J Bone Joint Surg.* 1959;41B:248-9.
2. Zucherman J, Hsu Ka, White A, Wynne G. Early results of spinal fusion using VSP system. *Spine* 1988;13(5): 570-79.
3. Inamdar DN, Alagopalan M, Shyam L, Devdoss S, Devdoss A. Posterior lumbar interbody fusion versus intertransverse fusion in the treatment of lumbar spondylolisthesis, *Journal of Orthop Surg.* 2006;14(1):21-6.
4. Kim NH, Lee JW. Anterior interbody fusion versus posterolateral fusion with transpedicular fixation for isthmic spondylolisthesis in adults. *Spine* 1999; 24:812-7.
5. Marchesi DG, Thalgot JS, Aebi M. Application and results of the AO internal fixation system in non-traumatic indication. *Spine.* 1991;16(3 suppl):162-9.
6. Pihlajamaki H, Bostman O, Ruuskanen M et al. Posterolateral lumbosacral fusion with transpedicular fixation: 63 consecutive cases followed for 4(2-6) years. *Acta Orthop. Scand* 1996;67:63-8.
7. Steffee AD, Brantigan JW. The variable screw placement spinal fixation system. *Spine*:18:1160-72
8. Daves SH, Myers DL. Complications of lumbar spinal fusion with transpedicular instrumentation. *Spine.* 1992;17(suppl):S184-89.
9. Esses SI, Sachs BL, Dreyzin V. Complication associated with the techniques of pedicle screw fixation. *Spine* 1993;18:2211-19.
10. Ohlin A, Kralsson M, Duppe H, Hasselius R, Johnell IR. Complications after transpedicular stabilization of spine: A survivorship analysis of 163 cases. *Spine.* 1994; 19(24):2274-79.
11. Pihlajamaki H, Myllynen P, Bostman O. Complication of transpedicular lumbosacral fixation for non-traumatic disorders *Bone Joint Surg* 1997;79-B:183-9.
12. Whitecloud TS, Butler JC, Cohen JL, Candelora PD. Complications with the variable spinal plating system. *Spine* 1988;14:472-75.
13. Roy-Camille R, Salliant G, Mazel C. Internal fixation of the lumbar spine with pedicle screw plating. *Clin Orthop.* 1986; 203:7-17.
14. Kim NH, Kim DJ. Anterior

- |  |   |  |
|--|---|--|
| 15. interbody fusion for spondylolisthesis. Orthopedics 1991;14:1069-76.   | 17. spinal fixation. Int. Orthop 2006, 17 June, DOI. 10.1007/s00264-006-4-0161.   | 19. lumbar spine Clin Orthop 1993:247:111-6.   |
| 16. Dawson EG, Lotysch M, Urist MR. Intertransverse process lumbar arthrodesis with autogenous bone graft. Clin Orthop.1981:154:90-96. | 18. Krag MH. Biomechanics of thoracolumbar fixation and fusion. A review. Spine.1991:16:S84-S99.  | 20. Chow SP, Leong JCY, MA A, Yau ACM: Anterior spinal fusion for deranged lumbar intervertebral discs-A review of 97 cases. Spine 1980:5:452-458. |
| 16. Butt MF, Munir Farooq, Mir B, Dhar AS et al. Management of unstable thoracolumbar spinal injuries by posterior short segment       | 18. Soini J, Laine T, Pohjolainen T, Hurri H, Alaranta H. Spondylodesis augmented by transpedicular fixation in the treatment of osteoarthritic and degenerative disorders of the | 20. Massie JB, Heller JG, Abitbol JJ, McPherson D, Grafin SR, Postoperative post spinal wound infection. Clin Orthop. 1992:284:99-108.             |

# Open Reduction and Internal Fixation of Intercondylar Fractures of Humerus in Elderly

Shafaat Rashid, M. A Halwai, B.A. Mir, Anwar, Nasir, Ab. Qayoom

## Abstract

We treated 20 patients at an average age of 74 years, range (61 to 90 years) with intercondylar fractures of the distal humerus by open reduction using transolecranon approach. We fixed all fractures by two reconstruction plates at right angles to each other as recommended by AO group.

There were 8 male and 12 female patients, 4 fractures were classified as C1, 10 as C2 and 6 as C3 as per Muller's classification. Patients were assessed at an average postoperative follow up of eighteen months.

The patient assessment was done on the scoring system of devised by Caja C.L and Moorani A' and were assessed as excellent 40%, good 35%, Fair 20 % and Poor 5%. The incidence of complications like wound infection nerve palsies, implant failure and pseudoarthrosis was not higher in these elderly patients. Old age is not a contraindication to open reduction and internal fixation, it is important to restore function.

JK-Practitioner 2007;14(2): 88-91

## INTRODUCTION

Fractures of the distal end of humerus are difficult to treat. Conservative treatment of distal intraarticular fractures of humerus usually result in loss of elbow movement and permanent disability. Any treatment which requires prolonged immobilization leads to loss of elbow function due to fibrous ankylosis of joint. We have, therefore, operated on all patients with distal intraarticular fractures of humerus to allow early postoperative movement of joint. Since there is no large series in elderly patient except that of John H, Rosso R' we have analyzed our experience to determine whether surgical techniques used in young patients could produce similar results in elderly.

## Patient and Method:

From January 2002 to January 2005, 50 patients with closed intercondylar fractures of distal humerus attended bone and joint surgery

Table:- 1

S. NO	Age/sex	Mode of injury	Type of Fracture.	Max.ROM Weeks	Interval. Bet.surgery &.union of fracture & osteotomy
1	76/M	Fall	C2	18	16
2	88/F	Fall	C1	16	17
3	62/F	Fall	C2	12	10
4	78/M	Fall	C2	16	16
5	75/F	Fall	C2	17	16
6	65/M	Fall	C1	14	12
7	68/M	Fall	C2	16	14
8	65/F	Fall	C2	16	14
9	80/F	Fall	C3	20	18
10	61/M	RTA	C3	20	18
11	72/F	Fall	C2	18	20
12	81/F	Fall	C2	14	16
13	63/M	Fall	C2	12	10
14	82/F	Fall	C2	16	16
15	68/F	Fall	C3	18	18
16	65/M	Fall	C3	17	16
17	62/F	RTA	C3	16	17
18	78/F	Fall	C1	10	12
19	85/M	Fall	C3	-	Non-union
20	68/F	Fall	C2	12	10

**Keywords:** Internal fixation, intracandylar fracture, humerus.

## Authors' affiliations:

Shafaat Rashid, M. A Halwai, B.A. Mir, Anwar, Nasir, Ab. Qayoom  
Government Hospital for Bone & Joint Surgery Srinagar 190005

## Accepted for Publication :

December 2006

## Correspondence:

Dr. M.A. Halwai  
Department of Orthopuedics  
Bone and Joint Hospital Srinagar-190005

Hospital and were treated by open reduction internal fixation. Twenty of these were more than 60 years and form the basis of this study. There were 8 male and 12 female patients, mean age at operation was 74 years (range 61-90 years). Mode of injury was fall in 18 patients and road traffic accident in 2 patients, fractures were classified as per AO classification Muller et.al. in C1, C2 and C3 types. There were 4 C1, 10 C2, and 6 C3 fractures, fifteen fractures affected right and five-left elbow (table I).

#### **Operative Technique :-**

The average interval between injury and operation was 7 days. Regional anesthesia was used in 6 and general in 14 patients. In all cases operation was performed under tourniquet with patients in prone position using AO technique, exposing fracture by a dorsal skin incision and chevron osteotomy of olecranon, apex of osteotomy was centered over trochlear notch. In all cases fracture was stabilized using two reconstruction plates at right angles to each other and a 4 mm cancellous transcondylar screw. Olecranon osteotomies were fixed by 6mm cancellous screw, reinforced by tension band wiring. In all cases crammer wire splint was used postoperatively as a protective shield until wounds healed. In all cases active ROM exercise were started from 2<sup>nd</sup> postoperative day.

**Review:** Patients were followed regularly for 12-36 months. Average follow up was 18 months, average healing time was observed as 14 weeks (range 10-20 weeks), maximum ROM was gained in an average time of 15 weeks (range from 12-20 weeks.)

Results were analyzed using the scoring system of Caja V.L, Moorani R. It is a 100 point scoring system developed to evaluate the results of surgical treatment of distal humeral fractures (table -II). The scoring system considered four parameters: pain, range of motion, radiological quality of surgical reduction and level of social or sporting activity as prior to injury. Pain is considered most important parameter and ranged from 40-0 points depending upon its absence or presence of rest pain. The range of motion was studied by measurement of elbow range of motion required for activities of daily living as described by Morrey et.al and varied from 30-0 points. For radiological analysis of surgical results, the post operative and final follow up radiographs were considered. The studied parameters are: -

1. Existence of articular step more than 1mm.
2. Existence of articular diastases more than 1mm.
3. Malalignment of carrying angle ( $15^\circ$  valgus) more than 10mm
4. Malalignment of trochlea capitellar angle ( $40^\circ$  ante version) more than  $10^\circ$ .
5. Articular surface reduction more than 1mm.
6. Presence of periarticular ectopic bone formation greater than 10mm.
7. Achievement of fracture union.

If none of these criteria are present 20 points are given, presence of any parameters reduce the score by 5 points and lack of fracture union scored 0 points.

Activity level was compared to activity before the accident; patients were given 10-0 points depending upon comparison with previous activity level.

#### **Result:-**

All fractures united in average time of 14 weeks.

Level of activity was higher in higher range of motion subgroup; severity of fracture affected the radiological functional and total score.

<b>Table II</b>		
Scoring system for the fractures of distal end of humerus		
A.	Clinical aspects:	Points
1.	No pain	40
2.	Activity related pain	
	a. occasional pain	30
	b. daily pain related to heavy activity	20
	c. daily pain related to light activity	10
3.	Rest pain	0
B.	Range of motion	
1.	No range of motion loss	30
2.	Loss of motion outside functional margins	20
3.	Flexion extension or pronation supination loss with in functional margins	10
4.	Flexion extension and pronation supination loss with in functional margins	0
C.	Radiological features :	
1.	articular surface step less than 1mm.	
2.	articular surface diastasis less than 1mm.	
3.	AP carrying angle malalignment more than $10^\circ$ .	
4.	Lateral trochlea capitellar angle ( $40^\circ$ ) malalignment less than $10^\circ$ .	
5.	articular surface reduction less than 1mm.	
6.	Paraarticular calcification less than 10mm.	
	<i>Maximum score 20 points , for each criteria not met 5 points are deducted . If pseudoarthrosis 0 points.</i>	
D.	Activity level :-	
1.	Level of activity as prior to injury	10 points
2.	Diminutions of previous level of activity	5 points
3.	Interruption of previous level of activity	0 point
	Excellent	90 to 100 points
	Good	70 to 85 points
	Fair	50 to 65 points
	Poor	Less than 50 points

Minor complications occurred in some patients but there was one non-union. Rest of fractures healed uneventfully although healing time was prolonged up to 18 weeks in 3 patients and 20 weeks in one patient. Screws backed out in 2 patients because of osteopenic bone. There was one ulnar nerve palsy because of a backed out screw, which was pressing upon the nerve, and it resolved with removal of the backed out screw, superficial wound infection occurred in 2 cases, which resolved with local wound care and antibiotics. Results were analyzed as excellent in 8 cases, good in 7, fair in 4 and poor in 1 patient because in this patient all screws loosened out and there was non-union at supra condylar region. This patient was advised a second surgery. He refused and was last in follow-up. Pain was absent in 12 cases, occasional in 5 and activity related in 3 patients. Range of motion was full in 6 patients, within functional range in 10 patients and below functional range in 4 cases.

Radiologically articular surface step more than 1mm was seen in 4 patients, articular surface diastases of more than 1mm in 1 patient, heterotrophic ossification more than 10mm in one patient. There was one case of pseudoarthrosis. Post surgical activity level was same as

prior to injury in 15 patients, diminished in 4 and was lost in one patient.

Table III

A.	Pain	No. of cases	%age
	No pain	12	60
	Occasional pain	5	25
	Activity related pain	3	15
B.	Range of motion (ROM)		
	Full ROM	6	30
	ROM more than functional range	10	50
	ROM less than functional range (functional range 30°-130°)	4	20
C.	Activity level		
	As prior to trauma	1	75
	Diminished	4	20
	Interrupted	1	5
D.	Radiological quality of surgical reduction.		
	Articular surface step >1mm	4	20
	Articular surface diastasis >1mm	1	5
	AP carrying angle malalignment >10°	1	5
	Heterotrophic ossification >10mm	1	5
	Pseudoarthrosis.	1	5
E.	Complications		
	Difficult reduction	4	20
	Ulnar neuritis	1	5
	Sup. Wound infection	2	10
	Screw back out	2	10
	Non union	1	5

**Result**

Excellent = 8(40%), Good = 7(35%), Fair = 4(20%), Poor = 1(5%)

**DISCUSSION:-**

The internal fixation of multifragmentary intra-articular fractures of distal humerus is a reconstructive dilemma especially if bone is osteoporotic. The superiority of open reduction internal fixation in young patients has been demonstrated in several studies but result in elderly patients are available in very few studies, Ansorge<sup>1</sup> has presented the results in individual cases. Where as encouraging results were seen in the study of John H. Rosso<sup>2</sup> also reported that age was not a factor in the outcome of these difficult fractures. Our study has proved it too. Rigid fixation and early range of motion are two prime factors to achieve best results in these fractures<sup>3,4,5</sup>.

We have fixed all our fractures with two 3.5 mm reconstruction plates at right angles to each other with a medial plate along medial column and a lateral plate on the posterior surface of lateral column. This type of construct has been found to be most stable both in bending and torsion<sup>6,7</sup>. Use of semi-tubular plates have often been found to fail whereas no reconstruction plate broke in the same studies<sup>8,9</sup>. Reconstruction plate also has advantage over DCP that it can be properly contoured to the complex anatomy of distal humerus.

We have fixed all olecranon osteotomies with 6.5mm cancellous screws and reinforced with tension band wiring. The pseudoarthrosis of olecranon osteotomies has been reported, independent of patients age in upto 30% of patients<sup>10</sup>. Except for one delayed union there was no major problem with olecranon osteotomies in our study, Use of chevron osteotomy has decreased the incidence of non-union<sup>11</sup>. Commonest problem encountered in our series were prominence of hardware and skin breakage over screw head, the incidence of non-union is low in these

Table IV

S. No	Pain	ROM	Activity	Articular surface Step > 1mm	Articular surface diastasis > malalignm Ent > 10°	AP Carrying angle	Heterotrophic ossification > 10mm	Pseudoarthrosis	complication	Result
1	No	>F.ROM	Full	Nil	Nil	Nil	Nil	Nil	Superficial Wound Infection	Excellent.
2	No	>F.ROM	-do-	-	-	-	-	-		Excellent
3	No	Full	-do-	-	-	-	-	-		Good
4	occasional	>F.ROM	-do-	-	-	-	-	-		Excellent
5	No	>F.ROM	-do-	-	-	-	-	-		Excellent
6	No	Full	-do-	-	-	-	-	-		Good
7	occasional	>F.ROM	-do-	-	-	-	-	-		Good
8	No	Full	-do-	-	-	-	-	-		Good
9	Activity Related	<F.ROM	Diminuation	+	-	-	-	-	Screw backout ulnar neuropathy	Fair
10	No	<F.ROM	Diminuation	+	-	-	-	-		Good
11	Activity Related	<F.ROM	Diminuation	+	-	-	-	-		Fair
12	occasional	>F.ROM	Full	-	-	-	-	-		Fair
13	No	Full	-do-	-	-	-	-	-		Excellent
14	occasional	>F.ROM	-do-	-	-	-	-	-		Good
15	No	>F.ROM	-do-	-	-	-	-	-		Good
16	No	>F.ROM	-do-	-	-	-	-	-		Good
17	No	>F.ROM	-do-	+	-	+	-	-		Fair
18	No	Full	-do-	-	-	-	-	-		Excellent
19	Activity related	<F.ROM	Interruption	+	+	-	+	Present	Non-union	Poor
20	No	Full	Full	-	-	-	-	-		Excellent

F= functional, ROM=range of motion, >=grater than, <=less than

fracture 2% and mostly occurs at supracondylar level<sup>11</sup>. In our series there was one non-union, because of severe osteoporosis all screws loosened out.

Incidence of ulnar nerve injury has been reported in 5-15% of patients<sup>12</sup>. In present series only one patient had ulnar nerve dysfunction because a backed out screw was

pressing on nerve, which resolved once screw was removed.

Old age is therefore not a contraindication to ORIF. Indeed in old people, it is most important to restore full function so that they can perform the activities of daily living and look after themselves.

## References:

1. Muller ME Allgower M: Technique of internal fixation of fractures. AO Manual of internal fixation 4<sup>th</sup> ed Spriger Verlag 2001.
2. Caja VL Moroni et.al: surgical treatment of bicondylar fractures of distal humerus. Injury 1994;25:433.
3. Cassebum WH :operative treatment of T or Y fractures of the distal humerus Am J surg 1952; 83:255
4. Ansoage D: Ergebnisse operative behandelte transcondylar Trummer brüche des Ellenbogengelenks im Erwachsenenalter. Zentralbl chir 1982; 107: 1096
5. Burri & Lob GH. Operative therapie der distalen humerus frakturen Hefte Unfallhelked, 1982, 155: 35
6. Jupiter JB: Intercondylar fractures of humerus JBJS 1994 76B:793.
7. Sanders R.A et.al :- Operative treatment of bicondylar intra-articular fractures of distal humerus .Clin orthop & related research 1993 292:26.
8. John H , Rosso R, : Operative management of distal humeral fractures in elderly. JBJS 1994 76B:793.
9. Morrey BF: A bio-mechanical study of normal function of elbow motion. JBJS 1981 63A:872.
10. Kaushal L, Singh SP: Comminuted intra-articular fractures of distal humerus int. orthop 1994 18:226.
11. John N Diana , Mathew L Ramsay: Decision making in complex fractures of distal humerus , current concepts and potential pitfalls Orthop Journa, 1985 11:12
12. Letsch R Schmit N : Intra-articular fractures of distal humerus Cline -Orthop 1989 241:238.
13. Helfert D L : bicondylar fractures of distal humerus in adults. Adv. Orthop. Surg. 1985, 81:223..
14. Henley MB and Perkere B: operative management of intra-articular fractures of distal humerus. J Orthop trauma 1987 8:223
15. Ackerman G and Jupiter JB: Non-union of fractures of the distal end of humerus. 1988; JBJS Am 70A, 75

# Incidence Of Hellp Syndrome In Severe Pregnancy Induced Hypertension And Its Impact On Maternal And Fetal Outcome:

Shafika Banoo, Tanuja Amin Makhdoom, Shahida Mir, Javid A Malik

## Abstract

We investigated the incidence of HELLP syndrome in patients with severe Pregnancy Induced Hypertension (PIH) and its impact on maternal and fetal outcome. It was a prospective study in which patients with severe PIH (B.P. > 160/110 mmHg) were investigated for HELLP syndrome in a tertiary care teaching hospital in Srinagar (J&K) and included one hundred consecutive patients who attended either out-patient clinic or emergency department. All the enrolled patients with severe PIH underwent detailed investigations including a complete haemogram, liver function tests and relevant investigations for hemolysis. We found that 10% patients had evidence of HELLP syndrome (i.e. Platelets < 100,000/mm<sup>3</sup>, Serum LDH > 600 IU/L, Serum SGPT > 70 IU/L and Schistocytes and Burr cells on peripheral blood film), whereas 8% had Partial HELLP syndrome (i.e. some but not all criteria of HELLP syndrome) and the remaining 82% had no evidence of HELLP syndrome. There was a significant difference in diastolic blood pressures among the three study groups ( $p < 0.05$ ). Cesarean delivery was indicated in 40% patients with HELLP syndrome. Though there was no maternal death in the present study, overall fetal mortality was 10% including 20% from patients with HELLP syndrome and 9.7% from those without HELLP syndrome. Disseminated intravascular coagulation (DIC) complicated 10%, acute renal failure 10% and placental abruption 20% patients of HELLP syndrome. We concluded that the incidence of HELLP syndrome in severe PIH is 10% and it increases with maternal age and gravidity. Partial-HELLP syndrome complicates 8% of such patients. Acute renal failure, DIC and placental abruption frequently occur in HELLP syndrome associated pregnancies. Perinatal outcome is also adversely affected and mortality may be as high 20%.

JK-Practitioner 2007;14(2): 92-94

## Introduction:

Although the association of hemolysis, elevated liver enzymes and low platelet count, the HELLP syndrome in pregnancy was recognized much earlier, it was first described in 1982. It is a frequent complication of severe pre-eclampsia-eclampsia that is characterized by multisystem involvement, dominated by microangiopathic hemolytic anemia, hepatic dysfunction and thrombocytopenia which usually progresses to disseminated intravascular coagulation (DIC). The HELLP syndrome in general complicates 0.2 - 0.6% of all pregnancies but its incidence increases to 4- 12% in severe pre-eclampsia.

The HELLP syndrome is usually misinterpreted as a non-obstetric problem because of its insidious onset and diverse clinical behaviour and thus the syndrome is often advanced before an accurate diagnosis is made. It may also present with convulsions, jaundice or bleeding from any mucosal surface and one-third of these patients present postpartum within 48 hours. It is common in white multiparous women between 25 and 37 years of age. We carried out a prospective study to know the incidence of this syndrome in severe in our population and to assess its impact on maternal and fetal outcome.

## Methodology:

Present study included one hundred consecutive patients with severe PIH (B.P. 160/110 mmHg) admitted to L.D. hospital of Government Medical College Srinagar India (a tertiary care institute) between July 1999 and December 2001. After written informed consent and clinical evaluation, haematological and biochemical investigations including platelet count, liver function tests and hemolytic profile were ordered in all the enrolled patients. We diagnosed HELLP syndrome if the platelet count was less than 100,000/mm<sup>3</sup>, serum glutamic oxalotransaminase (SGPT) more than 70 IU/L, serum lactate dehydrogenase (LDH) 600 IU/L and peripheral blood smear revealed evidence of microangiopathic hemolysis (i.e. Schistocytes and burr cells). All patients were investigated for renal failure, DIC, haemorrhage elsewhere (hepatic subcapsular, intracranial, etc) and placental abruption. Enrolled patients were divided into three groups, depending upon

## Authors' affiliations:

Shafika Banoo, Tanuja Amin  
Makhdoom, Prof. Shahida Mir,  
Department of obstetrics &  
Gynaecology  
Javid Ahmad Malik  
Hospital for Chest diseases  
Govt. Medical College Srinagar

## Accepted for Publication :

November 2006

## Correspondence:

Dr. Tanuja Amin Makhdoom  
Department of Obstetrics &  
Gynaecology Lalded Hospital  
Srinagar.  
E-mail: JAVAIDMALIK2009@Yahoo.co.in

**Key words:** Hemolysis, Hypertension in pregnancy, Elevated liver enzymes, Thrombocytopenia, Pre-eclampsia.

whether they had no feature of HELLP syndrome (Group A), some but not all the features (Group B) and those who fulfilled all the diagnostic criteria for HELLP syndrome (Group C). Treatment plan was standardized in all the three study groups and drugs known to cause hepatitis, hemolysis and thrombocytopenia were not used. Maternal and fetal outcome was assessed in terms of morbidity and mortality.

### Results:

Out of 100 enrolled patients with severe PIH, 82 had no investigation suggestive of HELLP syndrome (Group A), 8 had some but not all the criteria (Group B) and the remaining 10 met all the criteria required for diagnosis of this syndrome (Group C). Overall, on presentation 50% patients had pedal edema, 33% had vomiting, 12% had convulsions and 3% had jaundice. In group C, 70% patients were aged between 31 and 42 years whereas 77% of patients in other two groups were below 30 years ( $p < 0.05$ ). Seventy percent of patients from group C presented before 32 weeks of gestation. Unlike systolic blood pressures, there was a significant difference ( $p < 0.05$ ) among the three groups in diastolic blood pressures (Table 1).

Table 1: Systolic and Diastolic blood pressures in three study groups:

Group	Number	SBP (mean)	p-value	DBP (mean)	p-value
A	82	166.5	>0.05	113.8	<0.05
B	08	169.5	>0.05	119.0	<0.05
C	10	170.0	>0.05	121.8	<0.05

Group A: Severe PIH but no HELLP syndrome  
Group B: Partial HELLP syndrome  
Group C: HELLP syndrome

SBP: Systolic blood pressure  
DBP: Diastolic blood pressure

Overall 15 patients had platelet count below one lakh, out of which 2, 3 and 10 were from groups A, B and C respectively ( $p < 0.05$ ). All the patients in group A had SGPT levels between 28 and 49 IU/L, whereas four in group B and all the ten in group C (Table 2) had these levels between 75 and 90 IU/L ( $p < 0.05$ ). The serum levels of LDH (Table 2)

### Serum SGPT in IU/L

Table 2: Serum SGPT, LDH and Platelet levels in three study groups:

Group	Number	SGPT (mean)	p-value	LDH (mean)	p-value	Platelets $< 10^5/\text{mm}^3$	P-value
A	82	36.7	<0.05	285.6	<0.05	Nil	<0.05
B	08	74.3	<0.05	507.1	<0.05	5 (62.5%)	<0.05
C	10	82.3	<0.05	619.2	<0.05	10 (100%)	<0.05

### Serum LDH in IU/L

Table 3: Complications, Maternal and Fetal Mortality:

Complication	Overall	In HELLP syndrome
Renal Failure	3 (3%)	1 (10%)
DIC	1 (1%)	1 (10%)
Placental Abruptio	4 (4%)	2 (20%)
Intracranial Haemorrhage	1 (1%)	Nil
Maternal Deaths	Nil	Nil
Fetal Deaths	10 (10%)	2 (20%)

### DIC: Disseminated Intravascular Coagulation

were elevated to  $> 600$  IU/L in 14 patients including four from group B and ten from group C which on subgroup analysis using ANOVA revealed significant difference ( $p < 0.05$ ). Peripheral blood film showed presence

of Burr cells in 12 patients (2 from group B and 10 from group C) and Schistocytes in 10 patients (all from group C). Acute renal failure complicated one (10%), DIC one (10%) and placental abruptio two (20%) patients of HELLP syndrome.

Cesarean delivery was required in 15 (18.2%) patients in group A, three (37.5%) in group B and four (40%) in group C. Though there was no maternal death in any study group (Table 3), there were ten fetal deaths including eight (9.7%) from group A and two (20%) from group C ( $p < 0.05$ ).

### Discussion:

The triad of hemolysis, elevated liver enzymes and low platelets, the HELLP syndrome has been considered variously as, a variant of pre-eclampsia, misdiagnosed pre-eclampsia, mild DIC, etc. Considering the adverse effect this entity has on maternal and fetal outcome, early diagnosis and proper management are crucial. We studied 100 consecutive patients with severe pregnancy induced hypertension (PIH), to find out the incidence of HELLP syndrome in such patients and its impact on maternal and fetal outcome.

The HELLP syndrome complicates 0.4% of all pregnancies and 4 to 12% of those with pre-eclampsia. In the present study 10% patients with severe PIH had evidence of HELLP syndrome, which does not differ from that reported by some previous authors<sup>1</sup>. However other investigators have found its incidence as high 20% in such patients<sup>2</sup>. We found increased incidence (70%) of HELLP syndrome among multigravida in higher age group (31 to 42 years), an observation made by previous authors as well<sup>3,4</sup>. Though reported at age as young as 14 years<sup>5</sup> all our patients diagnosed as HELLP syndrome were between 22 and 40 years.

Due to lack of symptom specificity HELLP syndrome can be easily missed on casual clinical assessment.

Majority of our patients with HELLP syndrome presented at gestational age of 24 to 36 weeks with nonspecific symptoms including fatigue, malaise, vomiting, abdominal pain, jaundice, edema and convulsions which does not differ from that already reported<sup>6</sup>. Thus careful examination and relevant investigations in a given clinical setting are of immense importance for early diagnosis and proper management of any hypertensive pregnant patient.

Various complications observed in the present study included DIC, acute renal failure and placental abruptio which are already known. However we did not encounter some other reported complications<sup>7</sup> like hepatic rupture, pulmonary edema, retinal detachment, disseminated fungal infections, etc. Fortunately we had no maternal deaths among HELLP syndrome patients, whereas maternal mortality figures of 4 to 25% have been reported previously<sup>8-10</sup> depending among various factors the severity of PIH, nature of complications and expertise to handle such patients. There is a progressive rise in maternal morbidity and mortality as the pregnancy moves from class-III to class-I, HELLP syndrome<sup>11</sup>. Reported perinatal mortality in HELLP syndrome ranges from 8 to 60% which does not differ significantly from what was observed

in the present study (20%).

In summary, we conclude that the incidence of HELLP syndrome is 10% in pregnancies complicated with severe PIH and it increases with maternal age and gravidity. Partial-HELLP syndrome complicates 8% of such patients. Acute renal failure, DIC and placental abruption tend to complicate these pregnancies frequently. Perinatal outcome

is also adversely affected and mortality may be as high 20%. Optimal obstetric and neonatal care improve maternal and perinatal outcome. We recommend that all pregnant women having nonspecific symptoms should have complete hemogram and liver function tests and if required hemolytic profile as well, irrespective of maternal blood pressure.

## References

1. Davey DA, MacGilliveray I. The classification and definition of hypertensive disorders of pregnancy. *Am J Obstet Gynecol* 1988; 158: 892.
2. Michael Greary "The HELLP Syndrome". *British Journal of Obstet. Gynecol* 1997; 104: 887-91.
3. Schorr-Lesnick B, Leborics E, Dworkin B, et al. Liver disease unique to pregnancy. *Am J Gastroenterol* 1991; 56(6): 659-70.
4. Maria-G-Van P, Gastaf A.D., Hanswolf, et al. High prevalence of haemostatic abnormalities in women with history of severe pre-eclampsia. *Am J Obstet Gynecol* 1999; 180: 1146-50.
5. Weinstein L. Syndrome of hemolysis, elevated liver enzyme and low platelet count: A severe consequence of hypertension in pregnancy. *Am J Obstet Gynecol* 1982; 142: 159.
6. Mackenna, J., Dover N.L., Brame R.G: Pre-eclampsia associated with haemolysis, elevated liver enzymes and low platelets- an obstetric emergency? *Obstet & Gynecol* 1983; 62: 751.
7. Greer, I.A., Cameron A.D., Walker J.J.: HELLP syndrome - pathological entity or technical inadequacy: *Am J Obstet Gynecol* 1985; 152: 133.
8. Baha M.S., Mark M.T., Adel E.L. et al. Maternal perinatal outcome associated with syndrome of haemolysis, elevated liver enzymes and low platelets in severe pre-eclampsia/eclampsia. *Am J Obstet Gynecol* 1986; 155: 501-5.
9. Moodley J., Pillay H.: The HELLP Syndrome in severe hypertension crisis of Pregnancy-does it exist? *S. Afr Med. J.* 1985; 67: 246.
10. Aslan H, Gul A, Cebeci A. Neonatal outcome in pregnancies after preterm delivery for HELLP syndrome. *Gynecol Obstet Invest* .2004; 58(2):96-9.
11. Goodlin R.C., Cotton D.B., Haesslein H.C. Severe edema-proteinuria-hypertension gestosis. *Am J Obstet Gynecol* 1978; 132: 595.
12. Thiagarajah S., Bourgeois F.J., Herbert G.M. et al. Thrombocytopenia in pre-eclampsia: associated abnormalities and management principles. *Am J Obstet Gynecol* 1984; 150: 1.
13. Goodlin R.C.: Beware the great imitator-Severe pre-eclampsia. *Contemp. Obst. & Gyn.* 1982; 20: 215.
14. Riddell J 4<sup>th</sup>, Chenoweth CE, Kauffman CA. Disseminated *Scedosporium apiospermum* infection in a previously healthy woman with HELLP syndrome. *Mycoses* 2004; 47(9-10): 442-6.
15. Weinstein L. Pre-eclampsia/eclampsia with haemolysis, elevated liver enzymes and thrombocytopenia. *Obstet & Gynecol* 1985; 66: 657.
16. Lopez-Llera H, Espinosa H, Deleon M.D., et al. Abnormal coagulation and fibrinolysis in eclampsia. A clinical and laboratory correlation study. *Am J Obstet Gynecol* 1976; 124: 681.
17. Romero Arauz JF, LaraGonzalez AL, Ramos Leon JC, et al. Maternal morbidity and mortality in HELLP syndrome. *Ginecol Obstet Mex.* 2001; 69: 189-93.

## control study

## control study

## control study

[illegible]

1. The first step in the process is to identify the problem or issue that needs to be addressed. This involves gathering information and understanding the context of the problem.

2. Once the problem is identified, the next step is to define the objectives and goals of the project. This helps to clarify what needs to be achieved and provides a clear direction for the team.

3. The third step is to develop a plan or strategy to address the problem. This involves breaking down the problem into smaller, manageable tasks and determining the resources needed to complete each task.

4. The fourth step is to implement the plan. This involves putting the strategy into action and monitoring progress to ensure that the project is on track.

5. The final step is to evaluate the results of the project. This involves assessing the outcomes against the objectives and goals and identifying any areas for improvement.

	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036	2037	2038	2039	2040	2041	2042	2043	2044	2045	2046	2047	2048	2049	2050	2051	2052	2053	2054	2055	2056	2057	2058	2059	2060	2061	2062	2063	2064	2065	2066	2067	2068	2069	2070	2071	2072	2073	2074	2075	2076	2077	2078	2079	2080	2081	2082	2083	2084	2085	2086	2087	2088	2089	2090	2091	2092	2093	2094	2095	2096	2097	2098	2099	2100	2101	2102	2103	2104	2105	2106	2107	2108	2109	2110	2111	2112	2113	2114	2115	2116	2117	2118	2119	2120	2121	2122	2123	2124	2125	2126	2127	2128	2129	2130	2131	2132	2133	2134	2135	2136	2137	2138	2139	2140	2141	2142	2143	2144	2145	2146	2147	2148	2149	2150	2151	2152	2153	2154	2155	2156	2157	2158	2159	2160	2161	2162	2163	2164	2165	2166	2167	2168	2169	2170	2171	2172	2173	2174	2175	2176	2177	2178	2179	2180	2181	2182	2183	2184	2185	2186	2187	2188	2189	2190	2191	2192	2193	2194	2195	2196	2197	2198	2199	2200	2201	2202	2203	2204	2205	2206	2207	2208	2209	2210	2211	2212	2213	2214	2215	2216	2217	2218	2219	2220	2221	2222	2223	2224	2225	2226	2227	2228	2229	2230	2231	2232	2233	2234	2235	2236	2237	2238	2239	2240	2241	2242	2243	2244	2245	2246	2247	2248	2249	2250	2251	2252	2253	2254	2255	2256	2257	2258	2259	2260	2261	2262	2263	2264	2265	2266	2267	2268	2269	2270	2271	2272	2273	2274	2275	2276	2277	2278	2279	2280	2281	2282	2283	2284	2285	2286	2287	2288	2289	2290	2291	2292	2293	2294	2295	2296	2297	2298	2299	2300	2301	2302	2303	2304	2305	2306	2307	2308	2309	2310	2311	2312	2313	2314	2315	2316	2317	2318	2319	2320	2321	2322	2323	2324	2325	2326	2327	2328	2329	2330	2331	2332	2333	2334	2335	2336	2337	2338	2339	2340	2341	2342	2343	2344	2345	2346	2347	2348	2349	2350	2351	2352	2353	2354	2355	2356	2357	2358	2359	2360	2361	2362	2363	2364	2365	2366	2367	2368	2369	2370	2371	2372	2373	2374	2375	2376	2377	2378	2379	2380	2381	2382	2383	2384	2385	2386	2387	2388	2389	2390	2391	2392	2393	2394	2395	2396	2397	2398	2399	2400	2401	2402	2403	2404	2405	2406	2407	2408	2409	2410	2411	2412	2413	2414	2415	2416	2417	2418	2419	2420	2421	2422	2423	2424	2425	2426	2427	2428	2429	2430	2431	2432	2433	2434	2435	2436	2437	2438	2439	2440	2441	2442	2443	2444	2445	2446	2447	2448	2449	2450	2451	2452	2453	2454	2455	2456	2457	2458	2459	2
--	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	---

[illegible]

1. **Introduction**  
 This report provides a detailed analysis of the project's progress, highlighting key achievements and challenges encountered during the implementation phase. The primary objective was to develop a robust system capable of handling complex data processing tasks efficiently.

2. **Project Overview**  
 The project aims to streamline the data management process by integrating various data sources and implementing advanced analytics. The scope includes data collection, storage, processing, and visualization.

3. **Methodology**  
 The methodology employed a combination of agile development practices and structured testing procedures. Regular communication and collaboration among team members were essential for the successful execution of the project.

4. **Results and Findings**  
 The initial phase of the project successfully identified the requirements and established a clear roadmap. The development team has made significant progress in implementing the core functionality, with several key milestones achieved.

5. **Conclusion**  
 The project has demonstrated the feasibility of the proposed solution and has provided valuable insights into the complexities of data management. The findings suggest that the system is well-positioned to meet the project's objectives.

6. **Recommendations**  
 Based on the findings, it is recommended that the project continue to focus on optimizing the system's performance and ensuring data security. Further research and development are needed to address the remaining challenges.

# THE UNIVERSITY OF CHICAGO

Period	Period	Period	Period
period Dec, 2000 to Nov, 2000	period Dec, 2000 to Nov, 2000	period Dec, 2000 to Nov, 2000	period Dec, 2000 to Nov, 2000

1. The first part of the document is a list of names and their corresponding page numbers. The names are listed in a single column, and the page numbers are listed in a single column to the right of the names. The names are: John Doe, Jane Smith, Bob Johnson, Alice Brown, and Charlie White. The page numbers are: 10, 20, 30, 40, and 50.

2. The second part of the document is a list of names and their corresponding page numbers. The names are listed in a single column, and the page numbers are listed in a single column to the right of the names. The names are: John Doe, Jane Smith, Bob Johnson, Alice Brown, and Charlie White. The page numbers are: 10, 20, 30, 40, and 50.

3. The third part of the document is a list of names and their corresponding page numbers. The names are listed in a single column, and the page numbers are listed in a single column to the right of the names. The names are: John Doe, Jane Smith, Bob Johnson, Alice Brown, and Charlie White. The page numbers are: 10, 20, 30, 40, and 50.

4. The fourth part of the document is a list of names and their corresponding page numbers. The names are listed in a single column, and the page numbers are listed in a single column to the right of the names. The names are: John Doe, Jane Smith, Bob Johnson, Alice Brown, and Charlie White. The page numbers are: 10, 20, 30, 40, and 50.

5. The fifth part of the document is a list of names and their corresponding page numbers. The names are listed in a single column, and the page numbers are listed in a single column to the right of the names. The names are: John Doe, Jane Smith, Bob Johnson, Alice Brown, and Charlie White. The page numbers are: 10, 20, 30, 40, and 50.

[illegible]

1. The first step in the process is to identify the problem or issue that needs to be addressed. This involves gathering information and understanding the context of the problem.

trial	log <sub>10</sub> Gysemocology	trial	log <sub>10</sub> Gysemocology	trial	log <sub>10</sub> Gysemocology	trial	log <sub>10</sub> Gysemocology
1	1.0	1	1.0	1	1.0	1	1.0
2	1.0	2	1.0	2	1.0	2	1.0
3	1.0	3	1.0	3	1.0	3	1.0
4	1.0	4	1.0	4	1.0	4	1.0
5	1.0	5	1.0	5	1.0	5	1.0
6	1.0	6	1.0	6	1.0	6	1.0
7	1.0	7	1.0	7	1.0	7	1.0
8	1.0	8	1.0	8	1.0	8	1.0
9	1.0	9	1.0	9	1.0	9	1.0
10	1.0	10	1.0	10	1.0	10	1.0
11	1.0	11	1.0	11	1.0	11	1.0
12	1.0	12	1.0	12	1.0	12	1.0
13	1.0	13	1.0	13	1.0	13	1.0
14	1.0	14	1.0	14	1.0	14	1.0
15	1.0	15	1.0	15	1.0	15	1.0
16	1.0	16	1.0	16	1.0	16	1.0
17	1.0	17	1.0	17	1.0	17	1.0
18	1.0	18	1.0	18	1.0	18	1.0
19	1.0	19	1.0	19	1.0	19	1.0
20	1.0	20	1.0	20	1.0	20	1.0
21	1.0	21	1.0	21	1.0	21	1.0
22	1.0	22	1.0	22	1.0	22	1.0
23	1.0	23	1.0	23	1.0	23	1.0
24	1.0	24	1.0	24	1.0	24	1.0
25	1.0	25	1.0	25	1.0	25	1.0
26	1.0	26	1.0	26	1.0	26	1.0
27	1.0	27	1.0	27	1.0	27	1.0
28	1.0	28	1.0	28	1.0	28	1.0
29	1.0	29	1.0	29	1.0	29	1.0
30	1.0	30	1.0	30	1.0	30	1.0
31	1.0	31	1.0	31	1.0	31	1.0
32	1.0	32	1.0	32	1.0	32	1.0
33	1.0	33	1.0	33	1.0	33	1.0
34	1.0	34	1.0	34	1.0	34	1.0
35	1.0	35	1.0	35	1.0	35	1.0
36	1.0	36	1.0	36	1.0	36	1.0
37	1.0	37	1.0	37	1.0	37	1.0
38	1.0	38	1.0	38	1.0	38	1.0
39	1.0	39	1.0	39	1.0	39	1.0
40	1.0	40	1.0	40	1.0	40	1.0
41	1.0	41	1.0	41	1.0	41	1.0
42	1.0	42	1.0	42	1.0	42	1.0
43	1.0	43	1.0	43	1.0	43	1.0
44	1.0	44	1.0	44	1.0	44	1.0
45	1.0	45	1.0	45	1.0	45	1.0
46	1.0	46	1.0	46	1.0	46	1.0
47	1.0	47	1.0	47	1.0	47	1.0
48	1.0	48	1.0	48	1.0	48	1.0
49	1.0	49	1.0	49	1.0	49	1.0
50	1.0	50	1.0	50	1.0	50	1.0
51	1.0	51	1.0	51	1.0	51	1.0
52	1.0	52	1.0	52	1.0	52	1.0
53	1.0	53	1.0	53	1.0	53	1.0
54	1.0	54					

1. **Introduction**  
 2. **Background**  
 3. **Methodology**  
 4. **Results**  
 5. **Discussion**  
 6. **Conclusion**  
 7. **References**  
 8. **Appendix**  
 9. **Index**  
 10. **Table of Contents**  
 11. **Abstract**  
 12. **Summary**  
 13. **Key Words**  
 14. **Keywords**  
 15. **Subject Headings**  
 16. **Classification**  
 17. **Indexing**  
 18. **Keywords**  
 19. **Subject Headings**  
 20. **Classification**  
 21. **Indexing**  
 22. **Keywords**  
 23. **Subject Headings**  
 24. **Classification**  
 25. **Indexing**  
 26. **Keywords**  
 27. **Subject Headings**  
 28. **Classification**  
 29. **Indexing**  
 30. **Keywords**  
 31. **Subject Headings**  
 32. **Classification**  
 33. **Indexing**  
 34. **Keywords**  
 35. **Subject Headings**  
 36. **Classification**  
 37. **Indexing**  
 38. **Keywords**  
 39. **Subject Headings**  
 40. **Classification**  
 41. **Indexing**  
 42. **Keywords**  
 43. **Subject Headings**  
 44. **Classification**  
 45. **Indexing**  
 46. **Keywords**  
 47. **Subject Headings**  
 48. **Classification**  
 49. **Indexing**  
 50. **Keywords**  
 51. **Subject Headings**  
 52. **Classification**  
 53. **Indexing**  
 54. **Keywords**  
 55. **Subject Headings**  
 56. **Classification**  
 57. **Indexing**  
 58. **Keywords**  
 59. **Subject Headings**  
 60. **Classification**  
 61. **Indexing**  
 62. **Keywords**  
 63. **Subject Headings**  
 64. **Classification**  
 65. **Indexing**  
 66. **Keywords**  
 67. **Subject Headings**  
 68. **Classification**  
 69. **Indexing**  
 70. **Keywords**  
 71. **Subject Headings**  
 72. **Classification**  
 73. **Indexing**  
 74. **Keywords**  
 75. **Subject Headings**  
 76. **Classification**  
 77. **Indexing**  
 78. **Keywords**  
 79. **Subject Headings**  
 80. **Classification**  
 81. **Indexing**  
 82. **Keywords**  
 83. **Subject Headings**  
 84. **Classification**  
 85. **Indexing**  
 86. **Keywords**  
 87. **Subject Headings**  
 88. **Classification**  
 89. **Indexing**  
 90. **Keywords**  
 91. **Subject Headings**  
 92. **Classification**  
 93. **Indexing**  
 94. **Keywords**  
 95. **Subject Headings**  
 96. **Classification**  
 97. **Indexing**  
 98. **Keywords**  
 99. **Subject Headings**  
 100. **Classification**  
 101. **Indexing**  
 102. **Keywords**  
 103. **Subject Headings**  
 104. **Classification**  
 105. **Indexing**  
 106. **Keywords**  
 107. **Subject Headings**  
 108. **Classification**  
 109. **Indexing**  
 110. **Keywords**  
 111. **Subject Headings**  
 112. **Classification**  
 113. **Indexing**  
 114. **Keywords**  
 115. **Subject Headings**  
 116. **Classification**  
 117. **Indexing**  
 118. **Keywords**  
 119. **Subject Headings**  
 120. **Classification**  
 121. **Indexing**  
 122. **Keywords**  
 123. **Subject Headings**  
 124. **Classification**  
 125. **Indexing**  
 126. **Keywords**  
 127. **Subject Headings**  
 128. **Classification**  
 129. **Indexing**  
 130. **Keywords**  
 131. **Subject Headings**  
 132. **Classification**  
 133. **Indexing**  
 134. **Keywords**  
 135. **Subject Headings**  
 136. **Classification**  
 137. **Indexing**  
 138. **Keywords**  
 139. **Subject Headings**  
 140. **Classification**  
 141. **Indexing**  
 142. **Keywords**  
 143. **Subject Headings**  
 144. **Classification**  
 145. **Indexing**  
 146. **Keywords**  
 147. **Subject Headings**  
 148. **Classification**  
 149. **Indexing**  
 150. **Keywords**  
 151. **Subject Headings**  
 152. **Classification**  
 153. **Indexing**  
 154. **Keywords**  
 155. **Subject Headings**  
 156. **Classification**  
 157. **Indexing**  
 158. **Keywords**  
 159. **Subject Headings**  
 160. **Classification**  
 161. **Indexing**  
 162. **Keywords**  
 163. **Subject Headings**  
 164. **Classification**  
 165. **Indexing**  
 166. **Keywords**  
 167. **Subject Headings**  
 168. **Classification**  
 169. **Indexing**  
 170. **Keywords**  
 171. **Subject Headings**  
 172. **Classification**  
 173. **Indexing**  
 174. **Keywords**  
 175. **Subject Headings**  
 176. **Classification**  
 177. **Indexing**  
 178. **Keywords**  
 179. **Subject Headings**  
 180. **Classification**  
 181. **Indexing**  
 182. **Keywords**  
 183. **Subject Headings**  
 184. **Classification**  
 185. **Indexing**  
 186. **Keywords**  
 187. **Subject Headings**  
 188. **Classification**  
 189. **Indexing**  
 190. **Keywords**  
 191. **Subject Headings**  
 192. **Classification**  
 193. **Indexing**  
 194. **Keywords**  
 195. **Subject Headings**  
 196. **Classification**  
 197. **Indexing**  
 198. **Keywords**  
 199. **Subject Headings**  
 200. **Classification**  
 201. **Indexing**  
 202. **Keywords**  
 203. **Subject Headings**  
 204. **Classification**  
 205. **Indexing**  
 206. **Keywords**  
 207. **Subject Headings**  
 208. **Classification**  
 209. **Indexing**  
 210. **Keywords**  
 211. **Subject Headings**  
 212. **Classification**  
 213. **Indexing**  
 214. **Keywords**  
 215. **Subject Headings**  
 216. **Classification**  
 217. **Indexing**  
 218. **Keywords**  
 219. **Subject Headings**  
 220. **Classification**  
 221. **Indexing**  
 222. **Keywords**  
 223. **Subject Headings**  
 224. **Classification**  
 225. **Indexing**  
 226. **Keywords**  
 227. **Subject Headings**  
 228. **Classification**  
 229. **Indexing**  
 230. **Keywords**  
 231. **Subject Headings**  
 232. **Classification**  
 233. **Indexing**  
 234. **Keywords**  
 235. **Subject Headings**  
 236. **Classification**  
 237. **Indexing**  
 238. **Keywords**  
 239. **Subject Headings**  
 240. **Classification**  
 241. **Indexing**  
 242. **Keywords**  
 243. **Subject Headings**  
 244. **Classification**  
 245. **Indexing**  
 246. **Keywords**  
 247. **Subject Headings**  
 248. **Classification**  
 249. **Indexing**  
 250. **Keywords**  
 251. **Subject Headings**

**Section C**

1. The first step in the process is to identify the problem or issue that needs to be addressed. This involves gathering information and understanding the context of the problem.

[illegible][illegible][illegible][illegible]

For Anticardiolipin Antibody, rest of 5ml of blood was allowed to clot at room temperature and the sera were separated by configuration and was tested for Anticardiolipin antibodies by ELISA technique.

**TABLE I**

Distribution of 50 cases with unexplained repeated spontaneous abortions and 50 controls according to age

Age (Years)	Cases No %	Controls No %
21-25	15-30%	18-36%
26-30	26-52%	30-60%
31-35	7-14%	2-4%
36-40	2-4%	Nil
Number of pregnancies		

**TABLE II**

Distribution of cases according to the number of pregnancy wastage

S.No.	No. of pregnancies wastage	Total No. of patients
1.	Two Pregnancy wastage	31 (62%)
2.	Three Pregnancy wastage	16 (32%)
3.	Four Pregnancy wastage	2 (4%)
4.	Five Pregnancy wastage	1 (2%)

**TABLE III**

Distribution of cases according to the number of normal Pregnancies in control group

S.No.	No. of Pregnancy	No
1.	Two normal pregnancy	41 (82%)
2.	Three normal pregnancy	9 (18%)
3.	Four normal pregnancy	nil

**TABLE V DETAILS OF APL POSITIVE PREGNANCIES IN OUR STUDY GROUP**

S.No.	Patient	Foetal Loss			Alive	Acl	LA	Present Pregnancy
		1 <sup>ST</sup> Trimester	2 <sup>ND</sup> Trimester	3 <sup>RD</sup> Trimester				
1.	G <sub>1</sub> P <sub>1</sub> A <sub>1</sub>	3	1 PE Remote From term	1	1	23.5 GPL Units	+	—
2.	G <sub>1</sub> P <sub>1</sub> A <sub>2</sub>	1	1	1(IUGR)	1	16GLP+ Units	+	Amm1/4 Months
3.	G <sub>1</sub> A <sub>1</sub>	4(1-Missed Abortion)	Nil	Nil	Nil	15 GPL Units	+	Terminated by Elective LSCS
4.	G <sub>1</sub> A <sub>1</sub>	2	1 PE Remote From term	Nil	Nil	Nil Units	+	Amm3 months
5.	G <sub>1</sub> P <sub>1</sub> A <sub>1</sub>	3	Nil	Nil	1	Nil	+	-----
6.	G <sub>1</sub> A <sub>1</sub>	3(1-Missed Abortion)	Nil	Nil	Nil	14 GPL Units	+	—
7.	G <sub>1</sub> A <sub>2</sub>	2	Nil	Nil	Nil	Nil	+	Amm7 Months
8.	G <sub>1</sub> P <sub>1</sub> A <sub>1</sub>	Nil	1 H/O Abruption	2 Last was IUD	Nil	12 GPL Units	-	—

G-Gravida.

ACA- Anticardiolipin Antibody.

PE Remote from Term-Pre-eclampsia Remote from term.

LSCS- Lower Segment caesarian section. A-Abortion.

IUGR- Intra uterine Growth Retardation.

P-Parity.

LA- Lupus Anticoagulant Antibody

IUD- Intra Uterine Death.

**TABLE IV**

Distribution of 50 cases with unexplained Repeated spontaneous abortions and 50 according to lupus anticoagulant LAC and ACA positively.

Positivity of antibodies	Study Group	Control Group
LAC (+ve)	7	0
LAC (-ve)	43	50
ACA (+ve)	5	0
ACA (-ve)	45	50
Fisher test for LAC = P<0.012		
Fisher test for ACA = A<0.056		

### DISCUSSION:

The present study suggests association between lupus anticoagulant, anticardiolipin antibodies and pregnancy wastages. An association between antiphospholipid antibodies and pregnancy wastage in otherwise totally asymptomatic patients has been recently reported. However previously attention was focused on the role of antiphospholipid antibodies in causing repeated abortions mainly in women with SLE. In our study ACA and LAC positively was associated with repeated abortions even in the absence of SLE.

Our findings indicate that antiphospholipid antibodies are relevant factor in repeated abortions and may explain a considerable percentage of unexplained miscarriages.

The pathogenetic mechanism of this clinical event is poorly understood. There is extensive thrombosis of placental vessels. Placenta also shows extensive infarcts. It has been suggested that these antibodies may react with phospholipids antigen in the placental infarcts and may result in reduced nutrient transport across the

placenta. It has been suggested that these antibodies may react with phospholipids antigen in the placental infarction and result in reduced nutrient transport across the placenta.

In addition, there is evidence that plasma containing lupus anticoagulant antibody inhibits the production of prostacyclin by vascular tissue. Plasma containing Lupus anticoagulant interacts with vascular endothelium and inhibits prostacyclin synthesis. Since prostacyclins have a physiologic role in pregnancy, their inhibition may have an adverse effect on pregnancy. Similarly the hypothetically different role of LAC or ACA in these mechanisms are not known, and both may in fact be merely only a serologic

epiphenomenon of more general immunologic disorders.

In our study group there were 8 patients showing positive APL test. These patients showed wide range of fetal losses 2-5 with the mean of 3.2.

In this group of APL positive patients, a total of 33 pregnancies occurred.

Out of these 33 pregnancies, 26 (78%) terminated prematurely and in which 18 (54%) losses occurred in 1<sup>st</sup> trimester and 4 (12%) each in 2<sup>nd</sup> and 3<sup>rd</sup> trimester.

In our study of remaining 7 (21%) pregnancies, 3 patients were already alive issues without treatment.

Rest of the four patients are under our surveillance. All the patients were put on low dose aspirin, out of which one patient attended antenatal clinic regularly and was kept

under intensive monitoring and surveillance throughout her pregnancy. She developed pregnancy induced hypertension in her last trimester and pregnancy was terminated favourably at 37 weeks of gestation by elective lower section caesarean section. The male baby born to this patient weighed 2.8 Kg with Apgar score 10/10 and no congenital anomaly.

One of our patients hailed from a very remote village and lost track of her very early in the antenatal period.

The remaining two patients are in the midst of their pregnancy and have crossed the critical period of gestation where they had suffered losses earlier.

## References

1. Anitha ; Jyothy A Somayajulu G; Reddy PP : Prevalence of Lupus anticoagulant in recurrent miscarriage. *Journal of Obst & Gynaec of India* 45:5:95
2. Bajpai Rakhi ; Antiphospholipid antibody syndrome in pregnancy, Hughes syndrome, An overview. *Asian Journal of Obst, & Gynaec*, 4:4:2000
3. Barbuei T, Cortelazzo S, Gall M et al . Antiphospholipid in early repeated abortions: a case controlled study . *Fertil Steril* 50:589:1998.
4. Loizou S, McCrean JD, Rudge AC, Reynolds R, Boylec Harris EN , Measurement of anticardiolipin antibodies by an enzyme linked immunosorbent assay ( ELISA) Standardization and quantitation of results. *Clin Exp Immunol* 62:738-745:1985.
5. Pankaj Desai, Reena Anand , Malini Desai , Deepti Modi A.P. A Syndrome and recurrent missed abortion and still birth *J. of Obstet and Gynaec of India* 8:28-30:1998
6. Pankaj Desai; Malini Desai , Deepti Modi , Reena Anand ; Effect of treatment on cases testing positive for APA , A prospective study. *J. of obstet and gynaec of India* 3:34-36:1999.
7. Szczepanski M, Baver A, Gardas A, Duchinvi T Antiphospholipid antibodies and liprotein (a) in women with recurrent Fetal loss *International J of obstet and Gynaec* 61:39-44:1998.
8. Unander AM, Norberg R, Lennart Hahn Arfors Leopold. Anticardiolipin antibodies and complement in ninety - nine Women with habitual Abortion. *Am J Obstet & Gynaec* 156: 144:1987.

## **Seminoma in Undesended Abdominal Testes in a Patient With Persistent Mullerian duct syndrome.**

M.Y.Wani, Nissar A. Wani, Mudassir H Rather, Bashir A Chechak, Tariq A Sheikh Mohammad Shafi Wani

### **Abstract**

We report a case of seminoma in undesended testis of a patient with persistent Mullerian duct syndrome. This 38 year old patient was subjected to exploration with a preoperative presumptive diagnosis of tumor in undesended, abdominal testes, made on the basis of a detailed history, clinical and radiological examination (USG & CT Abdomen). On exploration a testicular tumor mass and an unrecognizable pelvic structure (subsequently proven to be uterus & Cervix) were removed and RPLND performed. Patient was subjected to adjuvant chemotherapy and had uneventful convalescence.

**JK-Practitioner 2007;14(2): 98-99**

### **Introduction:**

Persistent Mullerian duct syndrome is a relatively rare inherited defect of sexual differentiation characterized by failure of regression of the Mullerian ducts in males. The affected individuals are phenotypically normal males but have a rudimentary uterus and fallopian tubes along with unilateral or bilateral undesended testes. This internal male pseudohermaphroditism is caused by a deficiency of Mullerian duct inhibitory factor.<sup>1</sup> A genetic mutation of chromosome 19 seems to be implicated in this condition.<sup>2</sup> Like other undesended testes these gonads are at increased risk of malignant transformation (7-35%) and a variety of germ cell tumors have been reported in association with this syndrome.<sup>3</sup> These patients often present with Cryptorchidism or an inguinal hernia or rarely may present as abdominal mass as a result of malignant transformation of undesended testes.

### **Case report**

#### **Authors' affiliations:**

M.Y.Wani, Nissar A. Wani, Mudassir H Rather, Bashir A Chechak Tariq A Sheikh Mohammad Shafi Wani  
Department of Surgery,  
Govt Medical College Srinagar  
Kashmir

#### **Accepted for Publication:**

December 2006

#### **Correspondence:**

Dr. Sheikh Tariq Ahmad  
Registrar, Department of Surgery,  
Govt Medical College Srinagar  
Kashmir (J&K) 190010



**Seminoma testes  
Reementary Uterus & falopum tube**

A 38 years old male patient, non-smoker, normotensive, married 16 years back, father of two children, presented to OPD of SMHS hospital with one month's history of progressively increasing swelling lower abdomen and vague lower abdominal discomfort. Patient was constipated for last fifteen days from the date of admission. He also complained of easy fatigability.

Examination revealed that the patient was mildly anemic (HB: 10.5 gm %) and has a scar of previous surgery over right inguinal area. On checking his previous medical records, it was revealed that the patient had

**Key words:** *Mullerian Duct, Seminoma, Pseudohermaphroditism*

been diagnosed as a case of bilateral undescended testes with right indirect inguinal hernia six years before this admission and he had undergone right inguinal hernia repair with right orchidectomy at that time. Patient had well developed penis but scrotum was underdeveloped. Abdominal examination revealed firm to hard, non tender, fixed swelling occupying hypogastrium and right iliac fossa. Routine Lab investigations were normal except for an Hb of 10.5gm%. Ultrasound examination revealed mass of mixed echogenicity about 9 x 10 cm, predominantly solid arising from pelvis. There was no ascitis, para-aortic or pelvic lymphadenopathy and liver metastasis.

Abdominal CT scan of the patient showed the presence of a second soft tissue swelling within the pelvis but no Para-aortic lymphadenopathy or ascitis. A preoperative presumptive diagnosis of testicular tumor in left undescended testes was made and patient was subjected to exploratory laparotomy.

At operation a globular, mobile mass of 9 x 10 cm (subsequently proved to be Seminoma) was removed along with the second soft tissue swelling (Subsequently proved to be uterus with fallopian tubes). Retroperitoneal lymph nodes were also found to be enlarged and a RPLND was simultaneously performed. Patient was subjected to adjuvant chemotherapy in the post - operative period. Patient experienced uneventful post-operative period.

#### Discussion:

The persistent Mullerian duct syndrome (PMDS) is a rare disorder. The exact incidence is not known. The term PMDS was originally used by Nelson in 1939 and since then about 142 cases have been reported till 1997.<sup>1</sup> PMDS is thought to be heterogeneous disorder occurring either as a result of deficiency of Mullerian inhibiting substance or failure of the tissues to respond to it due to receptor or post receptor defect. The Mullerian inhibiting substance gene was cloned in 1986 and localized on the short arm of chromosome 19(19p 13.3).<sup>2,4</sup> The condition may occur sporadically or be inherited as an X-linked recessive or sex limited AR trait.<sup>1</sup>

Patient with PMDS have 46XY karyotype, male phenotypic development and in addition to testes, have Mullerian duct structures. Typically these phenotypic males have unilateral or bilateral undescended testes, bilateral fallopian tubes, a uterus and an upper vagina draining into prostatic utricle. The condition is commonly diagnosed

when Mullerian tissue is encountered during inguinal herniorrhaphy or orchidopexy, but at times may remain undiagnosed until 3<sup>rd</sup> / 4<sup>th</sup> decade of life when the patient presents with an abdominal mass as a result of malignant transformation of one or both undescended testes.

The PMDS can be divided into three groups:<sup>4</sup>

I. In 60-70% the testes with Vas deferens along with the uterus and fallopian tubes are located in the abdomen, in positions analogous to the ovaries. Inguinal hernia is not present in this group.

II. In 20-30%, one testis is found in the inguinal hernial sac or scrotum in association with contra-lateral inguinal hernia called hernia uteri inguinalis, with the other testes, uterus and fallopian tubes as its contents.

III. The remaining 10% patients have transverse testicular ectopia, where both testes are found in the same hernial sac with uterus and corresponding tubes.

The risk of malignancy associated with undescended testis in a case of PMDS is same as that of an abdominal testis 7-35% (avg. 18%). The commonest tumor reported in such cases is seminoma though other germ cell tumors have been reported. Bilateral seminoma in the settings of PMDS has also been reported.<sup>3</sup> Malignancies in uterus and vagina have not been reported. Most of the patients with PMDS are infertile, although fertility has been documented in some cases.

The semen analysis of infertile males reveals azoospermia or oligospermia. Management of PMDS by and large aims at preserving the potential fertility and minimizing of risk of malignant transformation of undescended testis. The Mullerian Structures which have no malignant potential should be left in place because the resection of uterus can damage the vasa deferentia which are embedded in the wall of the uterus. Thus leaving the Mullerian duct structures alone and performing staged or primary orchidopexy has been suggested but a long term follow up is then mandatory.<sup>5</sup> However, orchidectomy with excision of Mullerian structures followed by hormonal replacement is the other safe option, especially when long term follow up is not possible.

If malignancy is associated with this syndrome, the staging and treatment policy should be similar to that for other testicular tumors.

#### References

1. Grumbach MM, Conte FA. Disorders of sex differentiation. In: Wilson JD, Foster DW, Kronenberg HM, Larsen PR, (eds). Williams textbook of endocrinology. 9th ed. Philadelphia: WB Saunders, 1998: 1303-426.
2. Berkmen, F.: Persistent mullerian duct syndrome with or without transverse testicular ectopia and testis tumours. Br J Urol 1997;79:122-6.
3. Williams, J. C., Merguerian, P. A., Schned, A. R. et al: Bilateral testicular carcinoma in situ in persistent mullerian duct syndrome: a case report and literature review. Urology. 1994;44: 595.
4. Cates RL, Mattaliano RJ, Hession C. Isolation of the bovine and human genes from Mullerian inhibiting substance and expression of the human gene in animal cell. Cell 1986;573-716.
5. Migeon CJ, Berkovitz GD, Brown TR. Sexual differentiation and ambiguity. In: Kappy MS, Blizzard RN, Migeon CJ, (eds). The diagnosis and treatment of endocrine disorders in childhood and adolescence. Baltimore: Williams & Wilkins 1994:573-716.
6. Clarnette TD, Sugita Y, Hutson JM. Genital anomalies in human and animal models reveal the mechanisms and hormones governing testicular descent. Br J Urol 1997;79: 99-112.
7. Duenas A, Saldivar C, Castellero C, Flores G, Martinez P, Jimenez M. A case of bilateral seminoma in the setting of persistent Mullerian duct syndrome. Rev Invest Clin 2001;53: 193-6.
8. De U, Ghosh S, Chakraborty BP. Persistent Mullerian duct syndrome: a case report. J Indian Med Assoc 2002; 100:527-8,531.

# Ellis Van Crevald Syndrome

Chachoo Javaid, Arshid Bhat, Tariq Bhat, Tasaduq Bhat, Ramzan Bhat, Ahmad Feroze, Sami Jan

## Abstract

A case of Ellis Van Crevald syndrome without cardiovascular anomalies is being reported for its rarity. The disease is an Autosomal Recessive disorder involving the tissues of ectodermal and mesodermal origin. The patient had an unusual association of Bronchial Asthma.

JK-Practitioner 2007;14(2): 100-101

### Introduction

Ellis Van Crevald Syndrome (Chondroectodermal Dysplasia) is an Autosomal Recessive disorder involving the tissues of ectodermal and mesodermal origin.

### Case Report:

A 19 year old girl presented to us as a case of second episode of exacerbation of bronchial asthma and was found to have short stature. On examination she had a height of 4 1/2 feet, upper and lower extremities were shortened out of the proportion of trunk. The US was 76.5 cm, LS was 60.5 cm arm span of 78 cm, high arched palate, large tongue, upper lip was attached to the gums by 3 frenular bands, abnormally shaped peg like dysplastic teeth, hypoplastic nails, syndactyly of little finger of both hands, 6 digits in both hands and feet, bilateral genu valgum and inversion of right foot. Her psychomotor and mental development was within normal limits. She had a normal menstrual history. A skeletal survey showed short and tubular long bones with clubbed ends. Both knees showed hypoplasia and irregularity of lateral portions of upper tibial epiphyses and dome shaped metaphysis leading to the valgus deformity. Her chest X Ray and ECG was normal. The Echocardiography Scan and TEE was also normal. Her Pulmonary Function Test showed obstructive pattern with PEFR of 250 lt/min and reversibility of 22% after bronchodilation with short acting 2 agonists. The case was diagnosed as Ellis Van Crevald Syndrome without cardiovascular anomalies. Hence this case report is being given for its rarity.

### Discussion:

This rare skeletal dysplasia was first described by Richard W. Ellis and Simon Van Crevald in 1940 who coined the term Chondroectodermal Dysplasia<sup>1</sup>. This rare genetic disorder is transmitted in an autosomal recessive mode. The parents are of normal stature and both are carriers of mutation. A higher incidence of this syndrome has been demonstrated in Amish Community of Lancaster, Pennsylvania and Arabs of Gaza Strip. The incidence has been estimated at 1 per 60,000 live births<sup>2</sup>.

Etiology: Recent studies have found 2 genes in head to head configuration along the chromosome 4. Mutations in EVC gene at 4p16{EVC1} is purported not to cause the syndrome while as the mutations in the other gene at 4p20{EVC2} are currently thought to be implicated. The cytogenetic techniques have shown segmental uniparental disomy of chromosome 4<sup>3</sup>.

### Clinical Features:

These include post axial polydactyly of hands and sometimes feet. The hands are short and broad. Polydactyly may be just extra soft tissue not adherent to skeleton and devoid of bone, cartilage, joint or tendon; or the digit may show duplication with components like bifid metacarpals or there may be complete digit formation with its own metacarpal and complete soft tissue. There is also short limbed dwarfism. The bony dysplasia is characterized by acro-mesomelia- relative shortening of distal and middle segments as opposed to proximal segments. Knock Knees develop with growth due to poor development of Tibia. The Spine is unaffected<sup>4</sup>. The systemic anomalies are as follows:

1. Oral: partial hare lip, defect in Alveolar ridge, neonatal teeth,

**Key words:** Chondroectodermal dysplasia autosomal recessive, genetic disorder

### **Authors' affiliations:**

Chachoo Javaid, Arshid Bhat,  
Tariq Bhat, Tasaduq Bhat, Ramzan  
Bhat, Feroze Ahmad,  
SKIMS Medical College Bemina  
Sami Jan

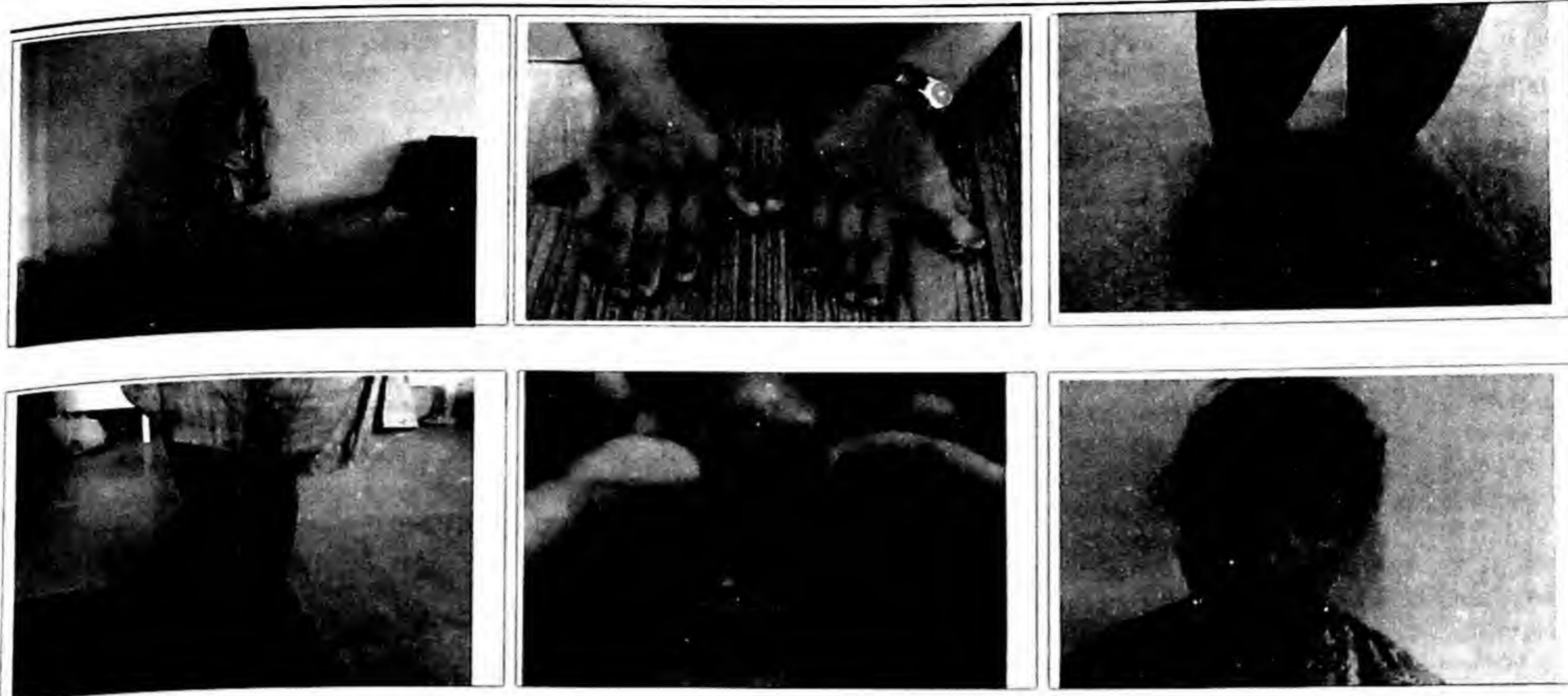
Department of Medicine  
Govt Medical College Srinagar  
Kashmir

### **Accepted for Publication :**

December 2006

### **Correspondence:**

Dr. Chachoo Javaid]  
Department of Medicine  
SKIMS, Medical College Bemina  
Email drjavaid-c@yahoo.com



delayed eruption, multiple frenula, abnormally shaped and microdontic teeth, congenitally missing teeth'.

2. Cardiac: cardiac anomalies are present in 50% of patients and include ASD, Single atrium or VSD'.
3. Thoracic parietal: Narrow chest, pectus carinatum, short and poorly developed ribs.
4. Genital: Epispadias, hypospadias, cryptorchidism'.
5. Limbs: Low Iliac wings, spur-like projections at mediolateral aspects of acetabula (trident pelvis), fusion of capitate and hamate, frequently a ninth carpal bone develop", short thickened tubular bones, talipes equinovarus, nail dysplasia.
6. Renal: Glomerulopathy with nephritic syndrome.
7. Hepatic: Paucity of bile ducts progressing to cirrhosis.
8. CNS: Dandy Walker malformation.

Anomalies of eye, mouth, palate and mental retardation have also been reported in the EVC syndrome. It may be associated with Hydrometrocolpos, Thymic Hypoplasia, Nephronophtosis. In our patient there was an unusual presentation of Bronchial Asthma which has not been reported so far for EVC Syndrome. Whether this is a mere coincidence or a rare manifestation of the same disease needs to be explored.

#### Diagnosis:

The condition can be diagnosed in the first trimester of pregnancy through a USG Scan looking for extra fingers of toes, cardiac defects, abnormalities of kidneys and underdeveloped limbs'. Clinical and radiological features help in diagnosing the case in children. Molecular genetic Testing is available in a few centers of the world.

The main differential diagnosis is Asphyxiating Thoracic Dystrophy (Jeune's Syndrome) . However, these two are different conditions and can be differentiated clinically'. Approximately 50% of all children with EVC Syndrome die in infancy due to breathing problems from the lack of chest development and associated heart anomalies. Significant Knee deformity can interfere with walking ability.

#### Treatment:

The disease requires a multidisciplinary approach to the treatment requiring surgical orthodontic and prosthodontic regimen to correct craniofacial morphology and teeth defects; distraction osteogenesis for limb lengthening; Corrective surgery for genu valgum; Pediatric Cardiologist at or soon after the birth; Urologist-if abnormalities of sexual organs or urinary tract are present; Clinical Geneticist and Respiratory Physician.

#### References

1. M.B.Popli, V.Popli; EVC; Ind J Radiol Imag, 2002, 1:4:549-550.
2. Faisal Haque, S.Amir Ahmad; Indian pediatrics 2004, 41; 867-868.
3. M.K.Mittal, P Jagmohan, N Sinha, B.B Thukral; Radiological Quiz-musculoskeletal, Ind J radiol Imag 2004, 14:217-218.
4. Springer S, Marino B et al : Mutations in a new gene in EVC Syndrome and Weyers'acrodental dysostosis, Nature genet 00, 24:283-286.
5. Varda and Ramos, chondroectodermal dysplasia-a case report, Eur J Orthod, 1996, 18:313-318.
6. T.W.S.Ray, C.G. Sanjeev, R. Venugopal, common atrium in child with EVC Syndrome, Heart 2002, 88:142
7. Ferns S, Biswal N, Rajkumar, Nalini P . EVC Syndrome, The Antiseptic, 2002, 99(i) 18-19.

# MRI in Joubert Syndrome

Naseer A Choh, Shabeer A Bhat, Suheel A Choh.

## Abstract

Joubert syndrome is characterized by inherited aplasia or hypoplasia of vermis with episodic hyperpnea and apnea, developmental delay, hypotonia, truncal ataxia, oculomotor, abnormalities and variable mental retardation. This clinical entity is probably underreported with a prevalence of less than 1 in 100,000.

**JK-Practitioner 2007;14(2): 102-103**

## Introduction

Joubert syndrome is prototype of congenital vermian hyperplasia (CVH); when other extracranial features are associated, this is referred to as CVH plus syndrome e.g. Meckel Gruber (with ectodermal dysplasia), senior-Loken (Leber congenital amaurosis plus juvenile nephronophthiasis), COACH (hepatic fibrosis), etc. We describe MRI features of an infant with seizure disorder and developmental delay which were typical of the classic Joubert syndrome.

## Case Report:

A 2 yr old child was referred for MRI of brain for seizure disorder and developmental delay. The child also had a history of episodic apneic spells (labelled as breath holding spells) and tongue protrusion. His physical examination was unremarkable.

MRI revealed absent cerebellar vermis with a narrow cleft separating the cerebellar hemispheres with a classical molar-tooth appearance of midbrain with prominent superior cerebellar peduncles. The fourth ventricle had an open-umbrella type appearance, connected with the median fissure separating the two cerebellar hemispheres.

Sagittal images revealed that fastigial point and primary fissure were lacking.

## Discussion:-

Joubert's syndrome was first described in five children with periodic hyperapnea, abnormal eye movements, ataxia and mental

retardation. Since then, approximately 200 cases of the disorder have been

reported, revealing a variable phenotype. Common signs and symptoms include hypotonia, impaired equilibrium and developmental delay. Associated abnormalities include characteristic facies with high rounded eyebrows and triangular shaped mouth, episodic hyperpnea or apnea



Fig 1. T1wt axial image shows median cleft separating the cerebellar hemispheres with open umbrella appearance of 4<sup>th</sup> ventricle.

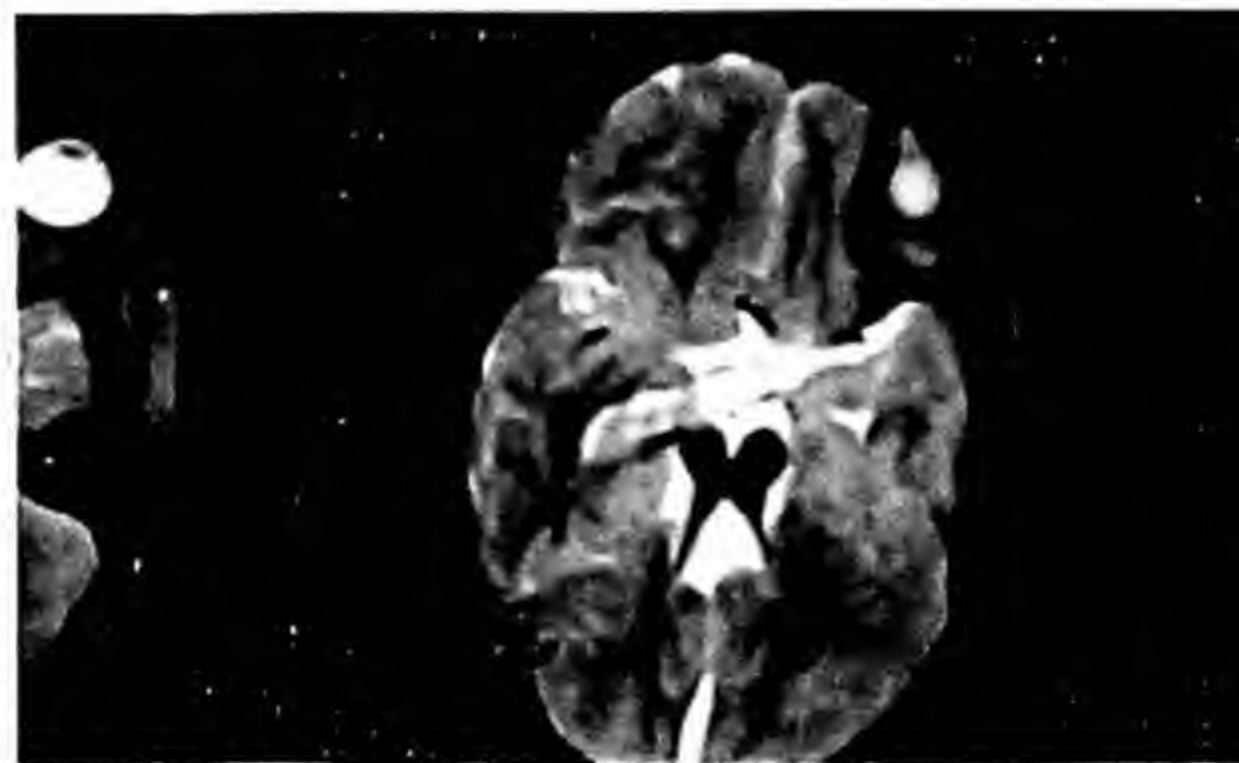


Fig 2. Molar tooth appearance of midbrain with thin superior cerebellar peduncles, visible due to superior vermian hypoplasia.

## Authors' affiliations:

Naseer A Choh, Shabeer A Bhat,

Department of Radiology,

Suheel A Choh.

Department of Pediatrics,

Govt Medical College Srinagar

Kashmir

## Accepted for Publication:

December 2006

## Correspondence:

Dr. Naseer A Choh

Department of Radiology,

Govt Medical College Srinagar

Kashmir (J&K) 190010

Email naseerchoh@rediffmail. Com.

in 50 to 75 % of patients , ocular abnormalities, renal and digital abnormalities .There are numerous "congenital vermian hypoplasia plus" eponymous syndromes like Meckel Gruber, senior-Loken, COACH syndrome, etc'.

The pathogenesis of Joubert syndrome involves cerebellar abnormalities such as vermian hypoplasia, neuronal loss and gliosis of purkinje layer, fragmentation of the dentate nucleus as well as malformations of brainstem, such as abnormal elongation of locus ceruleus, inferior olivary dysplasia, absence of pyramidal decussation, and anomalies of the dorsal column nuclei, solitary and trigeminal tracts. These abnormalities may account for the abnormal saccadic eye movements and abnormal respiratory pattern in Joubert syndrome"

Quisling et al' while reviewing the MR features of 35 patients with Joubert malformation, have described following morphologic features:

1. Dysgenesis of the isthemic portion of the brainstem at the pontomesencephalic junction.
2. Abnormally thick superior cerebellar peduncles perpendicular to brainstem.
3. Hypoplasia of cerebellar vermis with consequent enlargement of 4th ventricle and rostral shift of fastigium.
4. Saggital vermian clefting.

Maria BL et al have studied 15 patients with a diagnosis of Joubert syndrome; all patients had history of episodic hyperpnea and apnea , 12 patients has impaired smooth pursuit and defects in initiation of saccades and

quick phases. Two of the most consistant radiologic features were absent or hypoplastic posterior cerebellar vermis , and deformed midbrain and pontomesencephalic junction, with a classical " molar tooth appearance". Yachnis AL et al have reviewed the neuropathologic abnormalities and histogenesis of Joubert syndrome'. Kendall B et al have reviewed CT features of 16 children with Joubert syndrome; 7 of these also underwent MRI examination. All these children were developmentally delayed, and many had neonatal breathing abnormalities, congenital retinal dystrophy and supranuclear ocular motor abnormalities". Romano S et al have described 13 patients with Joubert anomaly , emphasizing the " molar tooth sign". Four patients in their series had dilated 4th ventricle and were less severely affected'. Alorainy I A et al have also described brainstem and cerebellar findings in isolated patients of Joubert syndrome'.

The cerebellar hemispheres have a normal volume. There is mild to moderate 4th ventricular enlargement with a bat-wing or open-umbrella appearance. Brainstem abnormalities observable on imaging are less common and include low position of the obex, elongation of the medulla, abnormally deep interpeduncular fossa and reduced olivary volume with enlarged superior ampulla of aqueduct. Supratentorial abnormalities include pituitary hypoplasia, mild prominence of subarachnoid spaces, mild lateral ventriculomegaly and delayed myelination as well as midline anomalies ( e.g. holoprosencephaly, frontonasal dysplasia, facial clefting )

## Bibliography:

1. Robertson R, Caruso PA, Trunit CL. Disorders of brain development. In. Scott W. Atlas Ed. Magnetic Resonance imaging of the brain and spine Vol. I, 3rd Ed. 324-325.
2. Osborn AJ. Diagnostic Imaging ; Brain I-1-34-37.
3. Yachnis A, Rorke L. Neuropathology of Joubert syndrome J Child Neurol 1999; 14 :655-659.
4. Quisling R, Barkovich AJ, Maria B. Magnetic Resonance imaging features and classification of central nervous system malformations in Joubert syndrome. J Child Neurol 1999; 14 : 628-63
5. Maria B, Hoang KB, Tusa RJ, et al. " Joubert syndrome " revisited : key ocular motor signs with magnetic resonance imaging correlation . J child Neurol 1999; 14 : 583-90.
6. Kendall B, Kingsley D, Lambert SR , et al Joubert Syndrome : a clinicoradiological study Neuroradiology 1990; 31(6): 502-6
7. Romano S, Boddaert N, Desguerre I, et al . Molar tooth sign and superior vermian dysplasia: a radiological, clinical and genetic study . Neuropediatrics 2006; 37(1): 42-5.
8. Alorainy 1A , Sabir S, Seidahmad MZ. Brainstem and cerebellar findings in joubert syndrome. J Comput Assist Tomogr 2006 ;30(1):116-21 Kossmehl , Daniela Grimm Rev Chir Orthop Reparatrice Appar Mot. 2005 May 91(3):267-71.

# Hydatid Disease of the Soft Tissues of the Lower Limb:

Mudassir Maqbool Wani, Abdul Munnon Durrani, Muhummed Shafi, Mubbashir Maqbool Wani, Mussadiq Khan.

## Abstract

A rare case of primary intramuscular hydatid cyst is reported here because of its unusual presentation. A young man presented clinically as having a soft cystic mass in the proximal region of the posterior compartment of the left calf. The clinical presentation showed a lesion with a predominantly cystic pattern, closely mimicking a soft-tissue neoplasm. Post operative histopathological examination confirmed hydatid cyst. A high level of awareness concerning the occurrence of these cysts is important, especially in regions where echinococcus is endemic. Radiological investigations can be helpful in non-invasive diagnosis of the condition. Surgical treatment follows the principles of malignant tumors, namely, wide surgical resection. A differential diagnosis of hydatid disease should be kept in mind in a case of swelling of the lower limb.

JK-Practitioner 2007;14(2): 104-106



Hydatidosis is known since Hippocrates. Echinococcosis or hydatid disease is a cosmopolite antrozoosis common to humans and several mammal species. It is endemic in many parts of the world where sheep and cattle are raised with the help of dogs. This disease is ubiquitously distributed with more frequent occurrence in the Mediterranean countries, East Africa, South

America, Russia and Australia'. Dogs are the principal host and sheep are the most common intermediate host. Dogs acquire the infestation by ingesting hydatid scolices from infected sheep organs. The parasite develops into a tapeworm in the dog intestine and tapeworm eggs are then excreted in dog feces and transmitted to humans via the fecal-oral route. The disease results from the development of the larval or hydatid form of the canine tenia (*Echinococcus granulosus*) in the organism. In humans the usual locations are the liver (52-77%), lung (8.5-44 %), abdominal cavity (8%), kidneys (7%), central nervous system (0.2-2.4%), and bone

(1-2.5%)'. The incidence of musculoskeletal hydatidosis is not clear. According to several authors, the incidence of musculoskeletal echinococcosis including involvement of subcutaneous tissue is 1% - 5.4% among all cases of hydatid disease'. However, a review of the English medical literature also revealed cases involving the musculature of the chest wall', the abdominal wall', the pectoralis major' sartorius' and sternomastoid muscles although it has been suggested that muscle provides a poor environment for the parasite because of the presence of lactic acid'.

A 35-year-old man had a two-month history of a painless, enlarging mass in the left proximal posterior part of leg. He had no history of fever but there was a history of prior trivial trauma two years back. A physical examination revealed a painless lesion, about six by eight centimeters, fixed to the deep tissue, with a smooth surface in the region of left gastrocnemius muscle. There was no redness, increased warmth or lymphadenopathy. The left hip and knee region had a full range of movement. Distal pulses were equal in both lower limbs. The remainder of the physical examination was unremarkable. Laboratory tests were done which included a complete blood count, liver function tests, kidney function tests, electrolytes, alkaline phosphatase, bilirubin and chest x ray, the results of all



## Authors' affiliations:

Mudassir Maqbool Wani, Abdul Munnon Durrani, Muhummed Shafi, Mubbashir Maqbool Wani, Mussadiq Khan.

Department of Surgery

Naseem-ul-Gani

Department of Orthopaedics

Government Medical College

Srinagar, J&K 190001 India

Accepted for Publication

March 2006

## Correspondence:

Mudassir Maqbool Wani,

PG Hostel, Room No.20,

Government Medical College

Karan Nagar Srinagar, J&K, India

190001.

E-mail: mudds\_123@yahoo.com

Ondutydoctor@yahoo.com

**Keywords:** Hydatid Gastrocnemius tapeworm, Echinococcus Granulosis

of which were normal. The USG of abdomen was normal. Based on clinical and radiographic data, the mass was believed to be a soft tissue tumor.

After a proper preoperative checkup, surgery was planned and done. The subfascial, extra muscular mass was exposed with posterior longitudinal incision. At operation there were found [picture 1 and 2] encapsulated, cystic cavity attached to the fascia of left gastrocnemius muscle, filled with 80ml of clear fluid and lined with a membrane and multiple daughter cysts. The cyst linings were excised, the cavity was thoroughly irrigated with hypertonic saline (5%) and the wound was closed. Histopathology confirmed the diagnosis of *Echinococcus granulosus* infestation. The patient was maintained on albendazole (800mg) daily for three months and remained on follow with no recurrence till date.

#### Discussion:-

Several factors would explain the exceptional nature of muscle localizations of hydatid cysts: efficacy of the hepatic and pulmonary barriers and muscle environment not favorable for growth of hydatid larvae. The predominant localization in the muscles of the lower limbs could be explained by the volume of the muscle mass and its rich blood supply. Differential diagnosis of a swelling of the calf is easy and most of the times the diagnosis is clinched with a proper investigation but preoperative diagnosis of musculoskeletal *E. granulosus* in calf difficult.<sup>10,11</sup> It may resemble any soft tissue tumor such as abscess, chronic hematoma, a synovial cyst, and necrotic malignant tumor.<sup>10,12</sup> Diagnostic modalities like ultrasonography, CAT Scan and MRI can make the pick up rate greater. Several patterns of disease have been recognized using various imaging techniques. These include the unilocular cyst, the multilocular lesion and the atypical complex or solid lesion.<sup>13</sup>

Ultrasonography is sensitive and useful in diagnosis, showing the size, localization and type of the cyst. CAT scan should be performed in suspicious cases or in order to determine the technique of surgery with demonstration of the relationship to adjacent organs. MRI is capable of adequately demonstrating most features of hydatid disease with the exception of calcifications.<sup>14</sup> Typical signs of

hydatidosis are multivesicular lesions with or without hypointense peripheral ring (rim sign)<sup>15</sup>. The diagnosis can be supplemented by specific IgG, complement fixation, indirect fluorescent, and ELISA tests<sup>16</sup>. The sensitivity of various serological tests used for hydatid disease varies from 64 to 87 %. The specificity of Casoni skin test is low because of this high 40% false positivity. ELISA is 80–100 % sensitive and 88–96% specific for liver cyst infestation, but less sensitive for lung (50–56 %) or other organ involvement (25–26 %). After surgical excision of the cyst, reagenic antibody (Ig E) titers decrease and become negative after 1–2 years. If titers do not decrease, recurrence of echinococcosis should be considered. Hydatid serology is only valuable when it is positive, negative serologic test does not exclude the diagnosis<sup>17</sup>. Management of the patient is complete surgical excision. If it isn't impossible to excise a large cyst en bloc, the cyst has to be drained intraoperatively, irrigated with a scolecidal agent such as hypertonic saline, and then excised. Excision is also recommended for those with a complication. The likelihood of recurrent infestation<sup>18</sup> is increased after rupture of the parent cyst; in addition leakage of cyst contents may cause anaphylactic shock<sup>19</sup>. Percutaneous needle biopsy is also not recommended because of the possibility of introducing scolices into the needle tract before surgical excision or biopsy and extirpation of cyst. If radical surgery is not possible, medical treatment of these patients does not eliminate the disease and new operations could be necessary.<sup>20</sup> A follow up with repeat ultrasounds of the abdomen and a complete physical examination would pick up those cases resistant to drugs<sup>21</sup>. This case illustrates that echinococcal disease should be considered in the differential diagnosis of every cystic mass in any anatomic location, especially when they occur in areas where the disease is endemic.<sup>22</sup>

Hydatid cysts are rarely found in muscles, even in highly endemic zones. The diagnosis must nevertheless be entertained depending on the clinical and endemic context. Ultrasound is the exploration tools of choice to confirm the diagnosis before surgery and avoid puncture. Exclusively surgical treatment is indicated, ideally for total pericystic resection without rupture.

## References

1. Echinococcosis/ Hydatidosis. In Cook G C and Zumla A. (eds). *Mansons Tropical Diseases* 21st ed Saunders, Edinburgh, 1 5 6 1 - 822.
2. Norman S Williams, RCG Russel, Christopher JK Bulstrode, Baileys and Love's short practice of surgery 24th ed
3. Ertan Bulbuloglu, Harun Ciralik, Murat Uzel, Kenan Coban, Bekir Borekci: Primary Subfascial Cyst Hydatid Disease in Proximal Thigh: An Unusual Localisation: A Case Report. *The Internet Journal of Surgery*. 2005. Volume 7 Number 1. Date 1 May 2006.
4. Alvarez-Sala, R., and Caballero, P.: E. cyst as a cause of chest wall tumor (Letter) and *Ann. thorac. Surg.* 43: 689, 1987.
5. Baig, M.A. Misgar, M.S., & Brijmojan, B: Primary E. cyst of the internal oblique muscle. *Int. Surg.* 60: 562, 1975
6. Abdel-Khaliq, R.A., & Othman, Y.: Hydatid cyst of pectoralis major Muscle, *Acta chir, Scand.* 152: 469, 1986.
7. Rask, M.R., & Latting, G.J: Primary intramuscular hydatidosis of the sartorius: Report of a case. *J Bone joint surg.* 52 A : 582, 1970
8. Surgical and medical management of rare echinococcosis of the extremities: Pre- and post-operative long-term chemotherapy Manfred Infanger, Peter Kossmehl, Daniela Grimm *Rev Chir Orthop Reparatrice Appar Mot.* 2 0 0 5 M a y 91(3):267-71.
9. Schantz, P.M. Editorial response: Treatment of cystic Echinococcosis. Improving but still limited. *Clin Infect Dis* 1999.29(2).310-311
10. Echinococcosis a differential diagnosis of calf swelling. *Schweiz Rundsch Med Prax.* 2005 Jan 26;





# Ovarian Hydatid Disease mimicking Ovarian Endometrioma.

Sajad A Malik, Malik Azad Hussain, Zahoor Gilkar, Ambreen Qureshi

A 35 year old woman presented to the infertility clinic as a case of primary infertility after five years of marriage. Both the husband and the wife were investigated for the cause of infertility. On transvaginal ultrasound left ovary was found to be cystic. The cyst was multiloculated with internal echoes. A provisional diagnosis of endometriosis with ovarian endometrioma (chocolate cyst) was made and laparoscopy planned. However the abdominal ultrasound showed a cystic lesion in the liver, which raised the suspicion of hydatid disease. Immunoelectrophoresis was performed and came out positive. Thus laparotomy was performed. The left ovary was found to be cystic with clear fluid in the cyst. Left ovariectomy was performed. The cyst in the liver was treated by cystectomy and omentoplasty. The histopathology of both the lesions confirmed echinococcal cysts. The post operative period was uneventful and the patient was put on post operative medical treatment. Till last follow up the patient was doing well.



Figure 2

Human Echinococcosis is a parasitic disease known since ancient times. It was first described by Hippocrates as a disease of the liver. However cysts have occasionally been reported in the spleen, kidney, skin, muscles and rarely in the heart, brain, ovaries and bones. The diagnosis of asymptomatic cysts depends on clinical suspicion. Ultrasonography and CT/MRI have almost 100% sensitivity and specificity and can reveal the morphology of the cyst. However Ovarian teratoma may also give the same sonographic picture as hydatid. Primary ovarian hydatid is very rarely reported (<1%). One such case has been reported by Adewunmi and Balingappa in a post menopausal woman who

was treated by surgical excision. A case similar to ours has been reported by Tampakoudis et al where there was a multicystic mass in the ovary and a similar mass in the liver which prompted a laparotomy and the histology confirmed the diagnosis. Thus like the other authors we are also of the opinion that Echinococcal disease should be considered in the differential diagnosis of pelvic masses especially in endemic areas and surgical treatment gives satisfactory outcome with low morbidity especially in cases with uncommon locations of hydatid cysts.

## Bibliography

1. Koukos E, Chatziantonio J, Chrissafis I, Anitsakis C Uncommon locations of hydatid cysts Singapore Med J 2007; 48(4): 119
2. Valle-Sanz YD, Lorento- Ramos RM. Sonographic and CT demonstration of hydatid cysts communicating with the biliary tree J Clin Ultrasound 2004; 32: 144-8
3. El Fortia M, Elhajaji E, Elmadani B, Khalil M Are they spherules of ovarian teratoma or daughter cysts of echinococcosis. Ultraschall Med. 2006; 27(6): 582-4
4. Adewunmi OA, Basilingappa HM. Primary ovarian hydatid disease in the Kingdom of Saudi Arabia Saudi Med J. 2004; 25(11): 1697-700
5. Tampakoudis P, Assimakopoulous E, Zafrakas M, Tzevelekis P Pelvic echinococcosis mimicking multicystic ovary. Ultrasound Obstet Gynecol 2003; 22(2): 196-8



Figure 1

JK-Practitioner 2007;14(2): 108

## Author's Affiliations :

Sajad A. Malik, Malik Azad Hussain, Zahoor Gilkar  
Department of Surgery  
SMHS Hospital, GMC, Srinagar.  
Ambreen Qureshi  
Department of Obstetrics and Gynaecology, Lal Ded Hospital, Srinagar Kashmir.

Accepted for Publication  
March 2007

## Correspondence :

Dr. Sajad A. Malik  
Department of Surgery  
SMHS Hospital, Srinagar.

# Extensive Lichenoid Drug Reaction in a Patient on Anti Tubercular Treatment

Qazi Masood Ahmad MD, Qazi Numan Masood MBBS; Iffat Hassan Shah MD, Farah Sameem MD

Lichen planus and drug induced lichenoid eruption are considered as distinct clinical and histopathological entities. Idiopathic lichen planus is commonly associated with conditions of altered immunity or chronic liver disease. On the other hand lichenoid drug eruption is temporally related to the intake of the implicated drug. The evolution of the rash is typical with pruritus followed by pityriasiform distribution of violaceous scaly plaques which have a tendency to coalesce and a characteristic histopathology. The appearance is psoriasiform, photosensitive distribution is seen. Mucosal involvement is the Post inflammatory hyper pigmentation is marked. We are reporting a case of extensive lichenoid drug reaction in a patient on antitubercular Treatment.

A sixty year old woman, an old case of treated lepromatous leprosy who developed Potts spine was put on anti tubercular treatment (ATT) three and a half months back. The patient presented to the Department of Dermatology with a one week history of generalized pruritus for which she was prescribed anti histaminics. Over the course of the next three weeks the patient developed purplish, pruritic scaly minimally indurated plaques and papules in a pityriasiform distribution on the trunk (Figure 1). The photodistribution on the face and hands was marked. The patient had involvement of lips, oral mucosa and genitalia. Scalp showed follicular lichenoid papules. Routine investigations were normal. Histopathological examination of a skin biopsy specimen of the lesion revealed a lichenoid histology. In view of the patient's Potts spine ATT was continued and topical steroids were prescribed.

Lichenoid drug eruption is known to be induced by Isoniazid and ethambutol<sup>1,2</sup> amongst the Anti Tubercular drugs. In contrast to Idiopathic lichen planus the lesion in lichenoid drug eruption are flatter, more confluent and psoriasiform. Photodistribution is marked with extensor involvement on limbs. The rash can arise anytime after the initiation of the drug therapy and even after discontinuation. At the same time the rash can subside even on continuation of therapy. Wickhams striae are not seen but post inflammatory hyper pigmentation is persistent.

In our patient the rash developed three and a half month after initiation of ATT. Her lesions were confluent and psoriasiform. Wickhams striae were absent and nail involvement was absent. Pruritus was intense and photodistribution marked. The patient had mucosal involvement. Her histopathology was suggestive of lichenoid drug eruption.

The patient was put on topical steroids and antihistaminics. Her ATT was not discontinued as the course of lichenoid drug rash is unpredictable and her clinical condition warranted continual ATT intake. On follow up after 3-4 months the patient's rash had subsided with intense post inflammatory hyper pigmentation.



JK-Practitioner 2007;14(2): 109

## Authors' affiliations:

Prof. Qazi Masood Ahmad, Iffat Hassan, Qazi Numan Masood, Farah Sameem

Department of Dermatology, STD and Leprosy  
Govt Medical College Srinagar  
India-190010

## Accepted for Publication :

January 2007

## Correspondence:

Dr. Qazi Masood Ahmad  
Professor and Head Department of Dermatology, STD and Leprosy  
Govt Medical College Karan Nagar  
Srinagar Kashmir (J&K) 190010  
E-mail: hassen\_iffat21@rediffmail.  
Com  
Mobile No: 9419079982

## References

1. Halery S, Shai A. Lichenoid drug eruption J AM Acad Dermatol 1993;29:249-55.
2. Grissmen ME, Warrenk, Mady A. Lichenoid eruption associated with ethambutol 1995;33:675-6
3. Van der Haute, Antoine JC, Lachapelle JM. Histopathological discriminating criteria between lichenoid drug eruption and idiopathic lichen planus: a retrospective study on selected samples Dermatologica 1989;199:10-13.

# Lateral Decubitus Position in Spinal Surgery - Current Concepts

Suhail Afzal MS, Asif Sultan MS, Mohd Iqbal MS, Shabir Ahmad Dhar

## Introduction:

Spinal surgeries were traditionally undertaken in the prone position for a long time. But some authors used to operate the spine in lateral position. The prone position has many anesthetic surgical disadvantages: difficult ventilation, compression of the endotracheal tube, and the presence of many pressure points. Eye ball compression has been documented to result in vagal stimulation, cardiac arrest, and even blindness. A catastrophic complication like cardiac arrest during surgery is likely to be fatal since resuscitation is often not possible in this position. Abdominal pressure is likely to result in profuse epidural bleeding and difficult ventilation. Lateral position during spinal surgeries has no disadvantage as mentioned above, but the surgeon has to have adequate training to operate the spine in lateral position. The purpose of this review article is to understand the current concept regarding the ideal position to operate the spine with a review of the author's own experience.

## Review:

Fourney DR et al<sup>1</sup> concluded from their study that they have performed a simultaneous anterior and posterior approach with the patients in the lateral decubitus position. They further stated that the simultaneous anterior-posterior approach is a safe and feasible alternative for the exposure of tumors of the thoracic and lumbar spine that involve both the anterior and posterior columns. Advantages of the approach include direct visualization of adjacent neurovascular structures, the ability to achieve complete resection of lesions involving all three columns simultaneously (optimizing hemostasis), and the ability to perform excellent dorsal and ventral stabilization in one operative session.

Dagher C et al<sup>2</sup> inferred that lumbar microdissection surgery is already performed under spinal anesthesia (SA) in many institutions. Following light sedation, SA is performed with the patient in the left lateral decubitus position, one to two levels above the herniated disc level. Isobaric 0.5% bupivacaine 3-3.5 ml was injected

intrathecally followed by wound infiltration with 15 ml of bupivacaine with 1/200 000 epinephrine prior to surgical incision.

Cybulski GR et al<sup>3</sup> used a modified lateral decubitus position with the scapula falling away from the side of exposure. This was used for T1-5 segment lesions, and a prone position was used for the (T-6)-(T-12) segment. Adequate decompression of the spinal canal was achieved in all cases. All patients who were ambulating preoperatively maintained ambulatory ability, and pain and/or further neurological improvement as well occurred in 75%.

Baulot E et al<sup>4</sup> performed thoracoscopy in the lateral decubitus position. The patient was prepared in the standard manner for a full thoracotomy. Surgical instruments that are needed for conversion to an open procedure must be in the operative room in the same position. Ventilation was stopped to the ipsilateral lung.

Sukegawa I et al<sup>5</sup> reported two cases of the rhabdomyolysis of the erector spinae muscles occurring after nephrectomy in lateral flexed decubitus position. A 39-year-old man (170-cm, 85-kg) underwent right nephrectomy for a right renal tumor. The patient was placed in a left flexed lateral decubitus position with a roll placed under the dependent iliac crest and upper half of the body was rotated backward for 6 h. Direct, prolonged pressure on the paravertebral muscle was the etiology of rhabdomyolysis in their cases. Although their cases were not severe and the complications were not induced, it must be kept in mind that excessive pressure in a limited area can damage the muscle during prolonged surgery.

Wawro et al<sup>6</sup> concluded from their study that the correction of posttraumatic kyphosis in the thoracolumbar region almost always requires a combined anterior and posterior approach because of the particular anatomic situation and the pathomorphologic changes. We suggest that the patient be placed in a right lateral decubitus position. This allows dual access to the spine by a posterior midline approach and a retroperitoneal thoracolumbar approach, so that simultaneous anterior and posterior manipulation, correction and stabilization of the spine are possible with no need to turn the patient intraoperatively.

Gonzalez Della Valle A et al<sup>7</sup> inferred from their study that the lateral decubitus position can cause dependent shoulder discomfort or result in traction on the brachial plexus. They measured pressure beneath the dependent shoulder and lateral angulation of the cervical spine in patients positioned in the lateral decubitus position for total hip replacement under epidural anesthesia. Inflatable pillows (Shoulder-Float) beneath the chest wall and head reduced pressure beneath the dependent shoulder from 66 to 12 mm Hg ( $P < 0.001$ ) and lateral angulation of the cervical spine from 14 degrees to 4 degrees ( $P < 0.001$ ). In a randomized crossover study of a further 15 patients, the use of inflatable pillows resulted in significantly less pressure beneath the dependent shoulder and chest wall than either a gel-pad or a 1000-mL lactated Ringer's bag. Inflatable pillows placed beneath the chest wall and head in the lateral

JK-Practitioner 2007;14(2):110-112

## Authors' affiliations

**Suhail Afzal**

Burjal Raddah, Rustaq Oman,

**Asif Sultan MS, Ortho**

Bone & Joint Hospital Srinagar Kashmir

**Mohd Iqbal, MS, Ortho**

Bone & Joint Hospital Srinagar Kashmir

**Shabir A Dhar,**

Bone & Joint Hospital Barzullah Kashmir India

## Correspondence

**Dr. Suhail Afzal**

P.O. BOX 427 BURJ AL RADDHA

POSTAL CODE 329

RUSTAQ OMAN.

e-mail: drsuhaila@yahoo.com haila@yahoo.com

Mobile: 0096895311945.

decubitus position provided lower pressure beneath the dependent shoulder than other support devices and facilitated alignment of the cervical spine. When patients lie on their side, this results in pressure beneath the shoulder and tilting of the head and neck to one side. These problems were effectively corrected with an inflatable pillow (Shoulder-Float).

Sato Ketal<sup>8</sup> concluded that hypotension after positioning is sometimes seen especially in patients with cervical spinal lesion operated on under prone position. Patients with spinal lesion and those with brain lesion are compared in the frequency of hypotension after positioning to prone. Sixty-one cases operated on with prone position were studied. Ages ranged from 40 to 82 (mean 61) years and ASA grade was 1 or 2 in each case. Cervical laminoplasty (group C) or craniotomy (group B) are performed in 40 and 21 patients, respectively. Ephedrine was administered when the systolic blood pressure decreased under 80 mmHg and the frequency of ephedrine use was compared. There were no differences in age and sex distribution between group C and B. The induction doses of propofol and fentanyl in group B were larger than those of group C, but ephedrine use in group C was more frequent than in group B. In T2-weighted image of the cervical cord, high signal intensity areas were depicted in cases with hypotension. The sympathetic flow descends in the medial part in the lateral funiculus. Damage of this pathway would cause autonomic dysfunction in patients with cervical spinal lesion and strict monitoring is necessary during positioning to prone.

Papin P et al<sup>9</sup> inferred that thoracoscopic release and fusion of the discs space followed in the same time by a posterior instrumentation and fusion is a good option. Six cases were done in the prone position, two in the lateral decubitus with shorter surgical time.

Chang SH et al<sup>10</sup> studied the incidence of perioperative ischemic optic neuropathy (POION) in spine surgery at our institution. POION is a rare but potentially devastating and untreatable complication of spine surgery, particularly that performed with the patient in the prone position. Anemia, hypotension, long duration of surgery, and significant intraoperative hydration may all be risk factors for this condition. All patients undergoing spine surgery should be informed about the low but definite risk of this condition, and every attempt should be made during surgery to maintain stable hemoglobin and mean arterial pressure and to avoid overhydration.

Sucato DJ et al<sup>11</sup> concluded that the results and

complications of patients undergoing a thoracoscopic anterior release and fusion comparing those performed prone with those in the lateral position. A thoracoscopic anterior spinal release and fusion in the prone position appears to achieve the same results as when performed in the lateral position for pediatric spinal deformity.

The authors own<sup>12,13,14</sup> experience is to perform operations like anterior instrumentation, posterior instrumentation, disectomy, anterior and posterior scoliosis correction, spondylolisthesis reduction in the lateral position with definite advantage to patient, anaesthesiologist, surgeon and other OT personnel with the best possible outcome.

## Conclusion:

Traditionally spine surgeries were undertaken in prone position which could be knee chest, kneeling, jack knife position etc. This additional gadgets like frames bolsters etc. This position has been accepted out of familiarity, training and experience. The problems faced by the surgeons including bleeding from excessive abdominal pressure resulting in epidural venous engorgement. Other disadvantages including static position during surgery and alteration position is almost impossible, combined anterior and posterior exposure not possible, maneuvering equipments like C-Arm image intensifier and operating microscope is difficult and strain on the surgeon.

The disadvantages to anaesthesiologists include the universal need for GA and catastrophic complications like cardio respiratory is almost impossible to manage.

The other OT personnel are at disadvantage because combined ant and posterior approach needs frequent change of position. The disadvantage to the patients include increased stress in small areas, cervical spine injury during position and high risk for elderly and obese patients. Many current articles support position of the patient in the lateral position with no disadvantages mentioned above. The patient can be positioned for combined anterior and posterior approach of the spine in lateral position. There is no bleeding, because there is less pressure occurring in the anterior abdominal wall and reduced venous engorgement. Anaesthetic risk is reduced in lateral position. Patient can be positioned into Kyphosis and Lordosis by just altering the hip and knee positions.

The authors own experience supports lateral decubitus position in all types of spinal surgeries with great advantage to the surgeon, anaesthesiologist, assistant, other OT personals and the patient.

## References:

1. Fournay DR, Abi-Said D, Rhines LD, Walsh GL, Lang FF, McCutcheon IE, Gokaslan ZL. Simultaneous anterior-posterior approach to the thoracic and lumbar spine for the radical resection of tumors followed by reconstruction and stabilization, *J Neurosurg*. 2001 Apr;94(2 Suppl):232-44.
2. Dagher C, Naccache N, Narchi P, Hage P, Antakly MC. Regional anesthesia for lumbar microdiscectomy: *J Med Liban*. 2002 Sep-Dec;50(5-6):206-10.
3. Cybulski GR, Stone JL, Opanami O. Spinal cord decompression via a modified costotransversectomy approach combined with posterior instrumentation for management of metastatic neoplasms of the thoracic spine. *Surg Neurol*. 1991 Apr;35(4):280-5.
4. Baulot E, Trouilloud P, Ragois P, Giroux EA, Grammont PM. Anterior spinal fusion by thoracoscopy. A non-traumatic technique: *Rev Chir Orthop Reparatrice Appar Mot*. 1997;83(3):203-9.
5. Sukegawa I, Miyabe M, Fujii T, Hoshi T, Takahashi S, Toyooka H. Rhabdomyolysis after nephrectomy in the lateral flexed decubitus position: *Masui*. 2003 Aug;52(8):882-5.
6. Wawro W, Boos N, Aebi M. Technique of surgical correction of post-traumatic kyphosis *Unfallchirurg*. 1992 Jan;95(1):41-6.
7. Gonzalez Della Valle A, Salonia-Razo P, Peterson MG, Salvati EA, Sharrack NE.

- Inflatable pillows as axillary support devices during surgery performed in the lateral decubitus position under epidural anesthesia. *Anesth Analg*. 2001 Nov;93(5):1338-43
8. Sato K, Kato M Hypotension after turning to the prone position Masui. 2003 Jan;52(1):46-8.
  9. Papin P, Arlet V, Marchesi D, Laberge JM, Aebi M Treatment of scoliosis in the adolescent by anterior release and vertebral arthrodesis under thoracoscopy. Preliminary results *Rev Chir Orthop Reparatrice Appar Mot*. 1998 May;84(3):231-8.
  10. Chang SH, Miller NR. The incidence of vision loss due to perioperative ischemic optic neuropathy associated with spine surgery: the Johns Hopkins Hospital Experience. *Spine*. 2005 Jun 1;30(11):1299-302
  11. Sucato DJ, Elerson E A comparison between the prone and lateral position for performing a thoracoscopic anterior release and fusion for pediatric spinal deformity. *Spine*. 2003 Sep 15;28(18):2176-80.
  12. Dr.P.Gopinathan et al : Lumbar Segmental Instability Treated by Expandable Spinal Spacer in PLIF. *J.Orthopaedics* 2004; 1(3)e4
  13. Dr.P.Gopinathan et al: Jacking up the spine A better way of treating lumbar spine instability, *J. Orthopaedics* 2005; 2(1)e3.
  14. Dr.P.Gopinathan et al lateral position the gold standard position in spinal surgeries *JCOA: Vol3, No.2 page 34-39*

# Doxycycline Induced Photo Onycholysis and Nail Pigmentation

Qazi Masood Ahmad, Iffat Hassan Shah, Qazi Nuaman Masood, Farah Sameem



A twenty five years old married female with a diagnosis of pelvic inflammatory disease was prescribed doxycycline 100mg twice daily. Fifteen days after starting treatment she noticed that all her finger and toe nails were getting discoloured a blackish blue hue at the same time she developed a reddish itchy rash on her face which was aggravated on exposure to sunlight. Cutaneous examination revealed that her finger and toenails were separated from the nail bed and had a blackish blue discoloration (Figure 1&2).

A diagnosis of photosensitivity with distal photo onycholysis was made and doxycycline was presumed to be the incriminating agent. The drug was discontinued and the nails reverted to their normal colour gradually.

Several drugs have been incriminated in the causation of photo-onycholysis including the tetracycline class of antibiotic.<sup>1-4</sup>

JK-Practitioner2006;14(2);113

## Authors' Affiliations :

Prof Qazi Masood Ahmad, Iffat Hassan Shah, Qazi Nuaman Masood, Farah Sameem

Department of Dermatology STD and Leprosy Government Medical College Srinagar

Email hassan-iffat21@rediffmail.com

## References:

1. Baran R, Dawber RPR, Richart B. Physical Sign. In Baran R, Dawber RPR, de Berker DAR, Haneke E, Tosti A, eds. Diseases of the Nails and their management, 3rd edn. Oxford: Blackwell Science, 2001; 86-103
2. Orentreich N, Harber LC, Tromovitch TH. Photosensitivity and photo onycholysis due to demethylchlortetracycline. Arch Dermatol 196; 83-730-7
3. Mallon E, Dawber RPR. Longitudinal melanonychia induced by minocycline. Br J Dermatol 1994; 130:794-5
4. Wise F, Sulzberger MB. Drug eruptions. Arch Dermatol Syphilol 1993; 24:549-67

# The Epidemiology of Fractures and Dislocations at District Hospital Kathua (J&K)

Rakesh Sharma MS Ortho, Liaqat Ali MBBS(Dip: Anaes:), Mukul Ubbot MBBS

## Abstract :

A study of 804 cases of fractures and dislocations was conducted. The aims were to study and analyse the pattern of fractures and dislocations, their age and sex distribution, cause of injury, rural and urban surrounding, site and side of injury, seasonal variation, associated injuries, percentage of simple and compound fractures and time gap between injury and hospitalization.

It was found that dislocations were relatively uncommon; upper limb injuries were more common than lower limb injuries and axial skeleton injuries. Radius and Tibia were the most commonly fractured bones followed by ulna and Femur.

A young adult male was found to be the person most at risk for an injury, and children particularly males were second age group most commonly involved.

The over all results showed that injuries due to fall accounted for 47.1 percent of the cases. More for limbs and skeleton, and their incidence increased for those over 40 years of age. Road Traffic accident caused the more serious fractures, such as those of the lower limbs. Right side was mostly involved in upper limb and lower limb fractures. Most of the compound fractures were seen in lower limbs. Incidence of fractures and dislocations was most in the months, of March, April and May.

**JK-Practitioner 2007;14(2):114-117**

## Introduction

The understanding of the Epidemiology of a disease is one of the method that helps to find out etiology of a disease and the factors that may lead to its control. The same way Epidemiology of fractures is of no less importance as Accidents in general and Road traffic accidents in particular remain a major community health problem.

Accidental injuries are a major cause of death in children aged over 1 year of age<sup>1</sup>, Road traffic accidents are the main cause of death in a developing country such as Saudi Arabia<sup>2,3</sup> and the 3rd most common cause of death in all age groups in developed countries such as the United States of America<sup>4</sup>

Apart from mortality, accidental fractures are probably one of the most reliable parameters of danger factors in lives of children<sup>5</sup> and adults.

The study is based on analysis of 804 cases of fractures and dislocations treated at District Hosp-Kathua. w.e.f April 1, 2004 to Sep 30, 2005.

## PATIENTS AND METHODES

The following data were collected age, sex, cause of injury, type of fracture its site (upper 3rd, middle 3rd, lower 3rd), side (Right, left) associated injuries, residence of patients (Rural or Urban), time gap between injury and hospitalization, seasonal occurrence and percentage of fractures requiring hospitalization. Cause of injury was divided into-Road Traffic Accidents which included those caused by Two wheelers, Four Wheeler and those caused to pedestrians. Falls which included slips and falls from height of more than and less than 5 feet. Assault which included sharp and blunt weapons and missiles and also those caused by Sports and Industrial machines or Agricultural implements.

The term 'type of fracture' used in this study signifies either simple (closed) fracture or compound (open) fracture. Associated injuries comprised Head injury, Chest injury, abdominal injury, vascular injury and Nerve injuries. Term multiple fractures was used if more than one fractures had occurred.

Time gap between injury and hospitalization was divided into less than 1 week, 1-3 weeks and more than 3 weeks.

Hospitalization of a patient meant those who required stay in Hospital for more than 24 hrs. and others were taken as OPD Patients. This study brought to light some very interesting points which are hardly recognised and realised.

**Key words:** fracture, dislocation, epidemiology

## Authors affiliations:

Rakesh Sharma, Liaqat Ali,  
Mukul Ubbot

District Hospital, Kathua, J&K, India

## Accepted for publication :

May 2006

## Correspondence

Rakesh Sharma (M.S. Ortho)  
239/15 Patel Nagar, Akhnoor Road,  
Talab Tillo, Jammu.  
Mob. 09419190019

**RESULTS**

A total number of 804 patients with 1026 confirmed cases of fracture or dislocation formed the basis of study. Of these patients 75.5 percent were males and 24.5 percent were females. Falls accounted 47.1 percent where as 36.0 percent of fractures and dislocations were due to Road Traffic Accidents.

There were 73.8 percent Simple fractures and dislocations and 26.2 percent Compound. Multiple fractures occurred in 27.6 percent of the cases. Associated injuries were encountered in 4.1 percent of the cases of fractures and dislocations. In limb fractures 52.8 percent occurred on right side and 47.2 percent on left side, 27.5 percent of upper limb fractures and 55.4 percent of lower limb fractures required admission in hospital for more than one day.

In upper limb long bones, the lower third was involved in 61.5 percent fractures, middle third in 27.7 percent and upper third was involved in 11.2 percent. Radius bone had maximum 69.3 percent involvement of lower third.

In lower limb long bones lower third was involved in 34.5 percent, middle third in 39.2 percent and upper third 26.3 percent. 23 cases of Epiphyse

al injury were seen, 21 were of lower radial epiphyseal injury and 2 of lower tibial epiphysis.

97.7 percent fractures and dislocations cases reported with in one week of injury and only 0.6 percent cases reported after 3 weeks of injury. Maximum cases had H/o Massages who reported late. Maximum number of fractures and dislocations were observed in the month of April and minimum number was seen in the month of November.

Over-all incidence among people from rural and urban background was almost equal. In urban people upper limb involvement was in 53.7 percent,

where as in rural people lower limb involvement was in 52.7 percent.

**FREQUENCY OF FRACTURES AND DISLOCATIONS**

Fractures accounted for 96.4 percent of the total

**TABLE I Summary of Findings in the commonest Upper limb Fractures**

	%of Total Fracture	Sex M : F	Residen R : U	Cause RTA : O	Side R : L	Type S : C	Site percentage			IAI Percent
							Upper 3rd	Middle 3rd	Lower 3rd	
Humerus	8.4	3.1:1	1.1:1	0.5:1	1.0:1	4.5:1	19.4	22.9	57.7	11.5
Radius	21.8	2.2:1	0.7:1	0.2:1	1.1:1	7.7:1	7.3	23.4	69.3	4.2
Ulna	11.2	5.9:1	0.9:1	0.3:1	1.1:1	8.8:1	12.5	39.9	47.5	8.3
Hand Bones	6.6	4.3:1	1:1	0.3:1	1.4:4	0.8 : 1	Thumb	Index	Others	
Clavicle	2.8	1.5:1	1.1:1	0.8:1	0.7:1	76 : -	Ray 11.6	Ray 23.2	65.2	4.4
							Medial 3rd 3.9	Middle 3rd 80.3	Lateral 3rd 15.8	1.3

R = Rural, U = Urban, O = Others, S = Simple, C = Compound, |A| = Incidence of associated injuries

**TABLE II Summary of Findings in the commonest Lower limb Fractures**

	%of Total Fracture	Sex M : F	Residen R : U	Cause RTA : O	Side R : L	Type S : C	Site percentage			IAI Percent
							Upper 3rd	Middle 3rd	Lower 3rd	
Femur	10.9	2.2:1	1.4:1	0.8:1	0.9:1	11.8:1	55.4	30.7	13.9	5.1
Tibia	17.8	4.7:1	1.2:1	1.1:1	1.3:1	2.0:1	16	41.2	42.8	7.5
Fibula	9.9	4.6:1	1.1:1	1.0:1	1.3:1	3.0:1	12.6	44.9	42.4	13.4
Foot Bones	4	5.4:1	0.6:1	0.6:1	1.0:1	3.2:1	Talus 4.6	Calcane 30.6	Others 64.8	0.9
Patella	0.9	1.9:1	0.6:1	0.9:1	1.1:1	11.5:1	Medial 3rd 3.9	Middle 3rd 13.0	Commin uted 57	4.3

R = Rural, U = Urban, O = Others, S = Simple, C = Compound, |A| = incidence of associated injuries

injuries. Upper limb bones were more frequently involved than those of the lower limbs and those of the axial skeleton.

The commonest ten fractures encountered in this study were those of the Radius, tibia, ulna, Femur, Fibula, Humerus, Hand bones, foot bones, clavicle and spine in order of frequency.

Table I and II summarizes the findings in the commonest upper and lower limb fractures and Table III summarizes the General findings in axial skeleton fractures.

In fractures and dislocations of vertebral Column, the age distribution has shown that the majority, 76.6 percent occurred in 20-50 years age group. While 2.3 percent occurred in those under 20 years age group and 20.7 percent in subjects of more than 50 years of age. Of these fractures 33 percent were complicated by spinal cord/root injury. Further analysis of the latter group showed no significant statistical difference in age, sex and site distribution but the evidence of fall as a cause was higher, accounting for 47.1 percent.

#### DISLOCATIONS

**TABLE III Summary of Findings in the Axial Skeletal Fractures**

	%of Total Fracture	Sex M : F	Residen R : U	Cause RTA : O	Type S : C	Site percentage			IAI Percent
						Cervical	Dorsa	ILumbar & Sacral	
Vertebra Column	10.9	2.2 : 1	1.4 : 1	0.8 : 1	11.8 : 1	55.4	30.7	13.9	5.1
Pelvis	2.3	3.5 : 1	2.2 : 1	63 : -	-	Ilium	Pubis Ulnum	Acetab	14.3
						14.3	80.9	4.8	

R = Rural, U = Urban, O = Others, S = Simple, C = Compound, |A| = incidence of associated injuries

Dislocations were uncommon, accounting only for 3.73 percent of the total fractures and dislocations. The Commonest dislocations encountered in this series were; dislocations of shoulder 35.2 percent, elbow 23.8 percent, small joints of hand and feet 20.9 percent, hip 12.4 percent and ankle 7.6 percent. Compound dislocations were encountered only in hand and ankle joints and accounted for 15.2 percent. Table IV. Summarizes the General finding of the commonest dislocations.

#### AGE AND SEX DISTRIBUTION

Age and sex distribution in this series showed a different pattern between males and females. 50 percent of the fractures and dislocations occurred in less than 30 years age group. All patients were grouped into three major groups, less than 20 years, 20-40 years and more than 40 years.

It was seen that less than 20 years of age accounted for 35.7 percent, 20 to 40 years of age 38.8 percent and more than 40 years of age accounted for 25.5 percent of the total fractures and dislocations.

In the case of females there was difference in this pattern, where less than 20 years age group accounted for 22.3 percent, 20-40 years accounted for 15.6 percent and more than 40 years age accounted for 35.7 percent.

The male age distribution showed that maximum injury occurred in young adults 20-40 years of age who accounted for 43 percent of the total males followed by less than 20 years age group, 36 percent and those more than 40 years accounted for 21 percent.

Although overall results showed that males outnumbered females by 4:1 the age related incidence showed a pattern in which the ratio was almost 3.5:1 in less than 20 years 5.6:1 in 20-40 years and 1.8:1 in more than 40 years age group.

#### MODE OF INJURY

In this series non-road traffic accidents were the major cause of injury accounting for 64 percent of the total, out of these 73 percent were caused by falls alone. Incidence of road traffic accident injury in relation to age was as follows. In children it accounted for 9.5 percent, while in

you  
n g  
adul  
t s  
the  
inci  
den  
c e  
incr  
ease  
d to  
2 2  
perc  
ent  
and

in those above 40 years of age accounted for 16 percent.

The sex/cause related incidence showed that females were less likely to be involved in road traffic accidents and this was the cause is only 8 percent of the total cases.

It was noted that road traffic accidents were a more frequent cause of injury in fractures of pelvis and lower limbs e.g in fractures of pelvis, Femur and leg, this was the cause in 68.3 percent, 46 percent and 52.4 percent respectively. Fall was the common mode of injury in bones and joints of upper limbs and spine e.g in case of Radius

**TABLE IV Summary of Findings in the commonest Dislocation**

	%of Total Dislocation	Sex M : F	Residen R : U	Cause RTA : O	Side R : L	Type S : C	IAI Percent
Shoulder	35.2	17.5:1	0.4 : 1	0.8 : 1	0.7:1	37 : -	2.7
Elbow	2.3	3.5 : 1	2.2 : 1	63: -	1 : 1	24 : 1	-
Hip	12.4	3.3 : 1	0.9 : 1	0.6 : 1	0.4 : 1	13 : -	-
Ankle	7.6	7 : 1	1.7 : 1	0.6 : 1	1.7 : 1	0.6 : 1	-
Others	21	3.4 : 1	0.6 : 1	0.3 : 1	0.6 : 1	1.4 : 1	-

R = Rural, U = Urban, O = Others, S = Simple, C = Compound, |A| = incidence of associated injuries

68.5 percent, ulna 61.7 percent and spine 55.8 percent. Of the dislocations 46.6 percent were caused by fall and 39 percent were caused by traffic accidents.

## DISCUSSION

The Epidemiology of fractures and dislocations in this study have confirmed the general impression that dislocations are very uncommon and that upper limb injuries are more frequent than those of the lower limbs and axial skeleton. In search for the bones most at risk of fracture, it was found that the radius and tibia are the most commonly involved followed by Ulna, Femur, Fibula, Humerus, Hand bones, Foot bones, Clavicle, Spine and Pelvis. Although fortunately fractures of spine are relatively uncommon accounting for only 2.8 percent of total injuries it was worrying to note that nearly 33 percent of these fractures were complicated by spinal cord/root injury and most of these fractures occurred in the young adult group, the financial, emotional and social burden must be immense<sup>6,7</sup>. Methods to minimize the risk of such an injury should be investigated.

By analysing the age and sex distribution in this series in search for the person at risk, it was found that most injuries occurred in young adult males (20-40 years), who accounted for almost 42 percent of the total patients.

Second commonest group to be involved was less than 20 years of age of both sexes which accounted for almost 35.7 percent. Females in general and adult females in particular were the lower risk group. This can be explained by the fact that vast majority of females in Kathua region are housewives and they spend little time outside their homes. More serious types of injuries were caused by blasts followed by road traffic accidents which accounted

for 1.8 percent and 36 percent respectively. In Kathua and Jammu road traffic accidents are a major community health problem. The advances in health care made the actual management more efficient, but there is a great need to work on preventive measures.

## CONCLUSION

The pattern of skeletal injuries in Kathua has shown that dislocations are uncommon, the radius and tibia are the most frequently fractured bones, a young adult male is the person most at risk of an injury and children of both sexes are the second age group most commonly involved. Research and implementation of methods to prevent and/or minimize these injuries is needed.

There is also need for improving the basic infrastructure at District level Hospitals so that number of Referrals is decreased.

Infrastructure inform of at least three Orthopaedic Surgeons at each Distt. Hospital where incidence of fracture is at least more than 500 fracture patients per year.

There should be fully equipped operation theater to deal with orthopaedic emergencies which includes: External fixators, Inter locking Nailing systems need less to say the routine plating and Nailing systems and last but not the least a Image intensifier.

There is need to have high way petrol and ambulances to shift the patient of Road Traffic mishaps which are frequently occurring on National Highway. These ambulances may be connected to Distt. Hospitals and Medical college through mobile phones.

There is a urgent need to set up a Trauma Center on National Highway especially at Kathua seeing the magnitude of Trauma.

## References

1. Sibert J.R. Maddocks G.B. and Brown B.M. Childhood accidents an endemic of epidemic proportion. Arch. Dis. Child. 1981; 56, 225.
2. Al. Qasabi Q. O., Mofarreh M.A., Al-Bunayan AR. et al. (1989); Road Traffic accident fatalities in Riyadh Central Hospital 1400-1407. (1980-1987) Ann. Saudi. Med. 9, 237.
3. Mufti M.H. Road traffic accidents on a Public Health Problem in Saudi Arabia. J. Traffic Med. 1983; 2, 65.
4. AHA publication 55-005-1 (1985); Leading Causes of death, United States 1982 estimate, heart facts. In: Instructors Manual for basic life support, 171.
5. Landin L. Fracture patterns in children. Acta Orthop. Scand. Suppl. 1983; 54, 1.
6. Khawaski M. I., Hafez YA., Chabra A et al. Spinal Cord injuries, proceedings of 5th Saudi Medical meeting, Riyadh, Saudi Arabia, 1980; p 589.
7. Shaheen M.AE., AA Sadr, N. Al-Khudairy, F.A Khan, A Masalem and N. Sabet., Pattern of accidental fractures and dislocations in Saudi Arabia. Injury. 1990; 21: 347-350;

# A SOCIOLOGICAL PERSPECTIVE ON KASHMIRI WOMEN IN MEDICAL PROFESSION

Shamshad, B. A. Dabla

The highest mountain ranges around Kashmir have until recent times greatly isolated it from rest of the world. This isolation has at all times exercised a decisive influence, not only on the history and people of Kashmir, but their traditions, day to day living with minimal effect from the outside world. But with the passage of time, the communication with outside world improved and with many invasions from outside world i.e. from north, south and west of Kashmir, the traditions too started changing in Kashmir and so was its impact on thinking of people and life style, etc.

Like in many parts of the world, the medical practice in Kashmir has undergone a sea change over the ages. Ayurveda, Rigveda, Siddha, Yoga and Naturopathy were mostly practised in ancient times. In the 14th century, Unani and Tibb systems of medicine were practised along with Ayurveda, the great physician who cured the king Zain-ul-Abideen was Sharya Bhatt. During Zain-ul-Abideen times, there were many hospitals in Srinagar and towns of Kashmir. Another physician Yavareshwar (1484 A.D.) was respected and honoured by the Kashmiris.

Practice of medicine was usually a hereditary profession. Writing about the practice of Hikmat in Srinagar city, Bates remarks: The son succeeds the father in his practice. The medical knowledge of Vedas and Hakims was based on herbs, Ayurveda and scraping acquaintance with the Greek system of medicine, or of a few nostrums that are handed down from sire to son.

The agriculturists were acquainted with medical properties of many herbs, which they used to cure certain diseases. There was also a class of men and women who eked-out their existence by applying leeches. The women generally presumed to know great many effective remedies which they shared with their neighbours. The people of Kashmir had considerable confidence in Hakims, who had great influence with even opulent and educated people (Lawrence, 1895). The knowledge of surgery was well nigh non-existent and barbers were traditionally doing the excision and boils, carbuncles etc. According to Lawrence 1895, one had to walk deep in the stinking slush when snow melted in spring to cross the roads. Due to cold climate, the incidence of infection is less, but once infection occurred, it took the shape of epidemics. Apart from these local factors, the attitude of the people was unhelpful. The Kashmiri people being very traditional, had a deep hold over the all kinds of superstitious beliefs. The spread of any disease was normally ascribed to the will of God; so instead of obtaining any medical aid and recourse to any preventive measure, the people generally went to priests and holy men who gave them charms and amulets to be tied on arms or washed with water and taken orally. Hindus felt elated when small pox came to their families, thinking Goddess 'Shetla' had visited their families and performed Pujas as remedial measures. Till late into 20th century, many Kashmiris were hesitant to get vaccinations done.

In the beginning of nineteenth century, the western education and Allopathic medical knowledge began to trickle in the valley after Christian missionaries came to Kashmir. People began to leave superstition and came to know about preventive medicine and opted for treatment by trained doctors rather than by holy men. Still for a long time, illiterate village folks remained submerged in superstition. The western medical practice was brought to the valley in the year 1864 by Dr. Elmslie, but there being no hospital in the valley at that time, he worked in the open and operated under trees. After his death, came Dr. Theodore Maxwell in 1872, who got land for hospital at Rustam Ghari at the foot hills of Shankracharya hill. The famous Neve Brothers, the British missionary doctors worked in this hospital and became a legend in their time. It was due to these two brothers Dr. Arther and Dr. Ernest F. Neve who brought modern surgery to the valley. It was because of Dr. Elmslie appeal that Miss Fanny Butler, MD qualified women from England in 1887 came to Kashmir and started to work among women in Kashmir. She was the member of Church of England Zanana Missionary Society. She worked successfully for nine years at Srinagar and then died in 1889. In the year 1890 Dr. E. Neve started the Leper hospital for the Government on the shores of Dal Lake and managed it for the state. By the year 1882-84, there was a Sadar Hospital in Srinagar, dispensaries at Maharaj Gang and Rainawari and branch dispensaries in all districts. Diamond Jubilee Zanana Hospital for women at Nawakadal was started by 1897 and Cottage Hospital for European people at the foot hills of Shankaracharya Hill by 1891. Church of England Zanana Missionary Society Hospital exclusively for women was established at Rainawari in 1908 and at Anantnag in the year 1909. Among the lady missionaries whose dedication to the cause of women's health earned approbation were, Miss Butler, a lady doctor, Miss Irene Petrie and Miss Robinson, trained nurses, "who laid down their lives on duty in the valley." Miss Kate Knowles was yet another noble doctor who earned deep affection for her services to the women of Srinagar.

JK-Practitioner 2007;14(2):118-121

Author's affiliation

Shamshad, PhD Scholar, Bashir Ahmad Dabla University of Kashmir, India

Correspondence Prof. Bashir Ahmad Dabla Head Department of Sociology & Social Work University of Kashmir

The other prominent European and Indian doctors working in Kashmir were Dr. A Mitra from Bengal, who was first Chief Medical Officer of Sadar Hospital in Srinagar. It was his untiring efforts, which helped in opening of the other hospitals in the state. Later on Col. Dhuni Chand was the CMO. A state of art X-ray plant was installed in Sadar Hospital by the year 1913-14 and a dental wing was opened in January 1930, a sanatorium for T.B. patients was started in Tangmarg by 1929.

The year 1940 was notable in the history of the Medical department of the State Government. Several important schemes to provide better health care facilities to the people of State were introduced. These schemes included construction of two large hospitals one each in Jammu and Srinagar comparable with best Medical Institution elsewhere. The laying of foundation stone ceremony of the Srinagar hospital was laid by Marquis of Linlithgow, the then Viceroy of India on October 15, 1940 and finally opened by another Viceroy Lord Wavell on 11th of October 1945.

The educational position of Kashmiri woman was backward. The education imparted to them was largely of religious character. Education in the valley was restricted to the upper classes and was meant for the boys only. In every 1000 of its female population, only one was literate in 1901 and three in 1921<sup>2</sup>. There were, some social and economic causes against the quicker and greater progress in education of woman. The reason for reluctance on the part of parents to send their daughters to school was that they were required to work in the house and help their mothers especially if there was a large family to look after and also, the parents reluctance was seen when they took away their daughters from the schools as soon as the later reached to a certain age. This tendency was encouraged by the custom of early marriage. The most important factor was the conservative attitude of the people and their general indifference to the education of females. It was the women of Kashmir, who more than the men, hindered progress on account of their ignorance and superstition. The economic reason in the way of women's education, included, the poverty of parents. They preferred to spend on the education of their sons rather than daughters which they considered more important from the economic point of view<sup>1</sup>. Parents also thought that education was not necessary for a girl, she has to function as a wife and a mother as well. Such domestic education she was supposed to get at home from her mother.

#### **Changes among the Kashmiri Women**

The position of women continued to remain the same for centuries. It is believed that the conditions in Kashmir in general and the Kashmiri women in particular started undergoing change from the early decades of the 20th century. Two major factors were responsible for the initiation of this broader and deeper change. First, at the endogenous level, the society was changing due to the emerging political consciousness, expanding modern education, spreading of cultural awareness, deteriorating economic conditions and large scale social awakening created a situation in which women felt concerned about their position, especially their role and status in and out of family. Second, at the exogenous level, there was a strong impact of the process of Neobremization which refers to the process of impact of the outside/alien cultures and other developments on the inhabitants of Kashmir<sup>4</sup>.

The western society encouraged women to come out of their homes to earn like men. Kashmiri women, like women in other places, too were being affected by these changes. The mass change among Kashmiri women started from early 20th century. While the upper class women responded early and fully to the changing processes, other women belonging to lower socio-economic groups responded the change very late.

The women were deprived of their rights, but reminded of their duties. But with the changing times, the role of women changed from only a child bearing and rearing machine to the bread earner too. Due to the rapid growth of industrialization and modernization, urbanization, development in the field of transport and communication, improvement in science and technology, not only new roles for the women emerged, but also new social norms and values developed. The women started getting education and showed interest in the political and professional fields. Besides, the other areas of social living, they began to come out of the domestic spheres and took professional roles outside their homes. This helped them to realize their potentialities, broaden their outlook and create a new meaning for themselves. The traditional role of a house wife gradually evolved into the dual and more fulfilling role of professional working wife and thus housewife, at the same time.

Thus the Kashmiri women have undergone a significant degree of change in the fields of education, profession, nutrition and health. Education including technical and professional education started now for women from primary to university level. They started to be employed in services, establishment, offices and professional and technical occupations<sup>5</sup>. They have now entered into occupations and professions formerly practised and dominated by men. Consequently, more and more Kashmiri women have not only stepped out of their traditional roles to slip into the world of work but have silently and surely made sizeable inroads into the traditional male bastion<sup>6</sup>. Education, economic liberalization has thrown open a new world of market opportunities for women which has played a great role in bringing the women of Kashmir, to adopt Medical Profession for the last more than two decades. Now women are awakened to their talents and realize the social responsibilities. Women are aware of the new trends and their own rights. The important reasons for their adopting the medical profession are their desire for higher status, economic self sufficiency utilization of individual talent, secure equality of status, self confidence, satisfaction, development of potential and personality. Besides, the educational status of parents has motivated the women to adopt medical profession. The changes in their positions and roles have brought along with them changing attitudes, changing behaviour patterns and the emerging problems related to medical profession. It is mostly through women's own talent, scientific knowledge and attitude, highly excellent academic

records and women's inclination and interest in modern professions including Medical Profession, that the Kashmiri women of today have reached the present climax.

Education played a decisive role in bringing the women of the valley to adopt medical profession. Gradually the professional education was started in the Kashmir, which was generally meant for boys only. Girls were encouraged to receive religious and general education and it was stated that the women were suitable for better performance of their domestic roles. This tendency changed altogether and consequently women received education in increasing number. The women of Kashmir developed an interest in medical subjects and thus acquired knowledge and finally got encouragement to get entry in medical profession. Also, the educational and economic status of the parents motivated the women of Kashmir to choose the medical profession. The other reasons were that the image of Medical Profession<sup>7</sup> and the opportunity to earn a large income in the Medical Profession motivated them towards it. Besides, utilization of the individual talent, aspirations for upward social mobility and the potential and desire to sense of competence motivated the women of Kashmir to enter into Medical Profession.

One of the important factors in motivating the women towards the medical profession was the fact that Medical Professionals were being respected and taken with the reverence by the Kashmiri society. The general awareness of the need of the health care for general women of the society made one feel to encourage the Kashmiri women towards the Medical Profession. The educational awareness among women led them understand their responsibilities towards society. Lately through media and other advertising agencies led the women of Kashmir society towards the Medical Profession. The parents began to think on the modern trends of professions also contributed to change their mind set and pulled them out of the traditional rigidity of keeping the women indoors and thus allowed their girl child to go for medical profession in and outside the state and also outside the country to achieve the medical degrees even higher degrees as well. Thus presently a favourable atmosphere of training the women for Medical Profession has been created. On the other hand the sense of responsibility among the women towards society has developed an economic awareness. The sense of being respected by the society is felt by the women medics at present.

In Kashmir valley, for a women to enter into medical profession was a distant dream. Some foreign medics were coming to Kashmir for practising their profession in some of the hospitals of the valley. Generally, the number of Kashmiri women medics was nil. Prior to the establishment of the Government Medical College Srinagar the state was undergoing a heavy expense in deputing 40-50 candidates to other medical colleges in India and had to grant heavy loans to them. The influence of modern education, increase in literacy and standard of living of people, and better communication contributed a lot towards it. The first Medical College in the state had its beginning in the year 1959. The first Medical College building was situated at Hazuri Bagh on the Banks of river Jhelum (The present LD Hospital). The first Principal of the Institution was Dr. G. V. S. Murti whose office was situated here as well. The basic Science Departments of the College were Anatomy, Physiology and Biochemistry were also situated here.

The present building of Government Medical College Srinagar was inaugurated by Mr. Bakshi Ghulam Mohammad, Prime Minister of J&K, on August 25, 1959, at Karan Nagar, which ultimately started functioning as full fledged Medical College and has produced thousands of medics till date. Not only medics with M.B;B.S degree but also every district hospital has postgraduate medics/medics manning the various specialties. The first batch of students admitted in the college was 67, 60 from J&K state and 7 from other states. The first batch of graduate students came out in 1964. The first women medics who qualified from Government Medical College Srinagar were Dr. Nayeema Akhter, Dr. Parveen Chesti and Dr. Bilques. After-wards Government Medical College, Jammu, SKIMS Medical College and ASCOMS, Jammu were established. Besides the Postgraduate Institute named as Sher-e-Kashmir Institute of Medical Sciences, Soura established in 1980 has added specialists and super-specialists in this profession. where the training for postgraduation in the medical sciences is imparted. For one or two decades since the inception of Medical College, a little number of Kashmiri women entered into medical profession. The women practitioners were less, and the women patients had to rely upon the male medics for the treatment of their disease. In early 1980's the female medics always would choose Obstetrics and Gynaecology as their career. However after 1985, as a result of change in Government policy, the number of students admitted to various medical colleges of Jammu and Kashmir State included 50% male and 50% females. This resulted in female medics opting for specialties other than Obstetrics and Gynaecology.

#### **Pioneer Kashmiri Women Achievers in the Medical Profession**

Now in our male dominated society, things have changed enormously. There is a technological revolution in the medical field<sup>8</sup>. The number, the quality and the skill of women doctors have increased in leaps and bounds, so that today, there are women doctors in every facet of medicine.

Myriads of women have served with distinction in the medical profession. Women doctors have become great administrators and academicians. They have excelled in Medical Research, Surgery, Gynaecology and Obstetrics, Forensic Medicine and also in the Social and Preventive Medicine. Women doctors are contributing excellent service to the society.

Along with men, the Kashmiri pandit ladies too started going for medical studies in late 40's. One of the first trained lady doctors is probably Dr. Prabha Labroo LMP followed by Dr. Sheela Razdan M.B;B.S 1948, Dr. Shanta Wazir Bhargava 1949, Prof. Dr. Gauri Bazaz Malik M.B;B.S, MD 1950, Dr. Prabha Ganju M.B;B.S 1952, Dr.

Durga Kaul Gariju M.B.B.S, 1954 and Dr. Shanta Raina. Most of these doctors were manning the Obstetrical part of the women folk and some of them performed minor gynae surgery as well. Inspite of less number of women doctors these pioneers have really looked after the women of Kashmir till better trained gynaecologists and obstetricians took over place of position in the Government Medical College, Srinagar. Dr. Shanta Wazir Bhargava and Dr. Gauri Bazaz Malik were both Pathologists who worked in Delhi only.

Some of the pioneer Kashmiri women medics who have made the name in the medical profession were:

Dr. Parveen Chisti (Pathology), Dr. Nayeema Akhtar (Pathology), Dr. Vimla Dhar (Pharmacology), Dr. Nirmal Raina (Pharmacology), Dr. Z. Jeelani (Pharmacology) Dr. Veena Amla (Pharmacology), Dr. Hamida Buch (ENT /Ophthal), Dr. Mahmooda Khan (Surgery), Dr. Pratiba Hak (Physiology HOD), Dr. Leela Choudhary, Dr. Sheela Kachroo (Obst. & Gynae), Dr. J. A. Naqashbandhi (Gynae and Obs), Dr. G. Dhar (Gynae and Obs) Pr. Dr. Wazira Khanam (Gynae and Obs) Pr. Dr. Bilques (Gynae and Obs) pr. Dr. Phoola Koul (Anaesthesiology) Pr. Dr. Chuni Mazzine (Anaesthesiology) Dr. H. Bingri became the Director of Health Service Kashmir, Dr. Sumagla Bhan (Paediatrician), Dr. Jyoti Kol

At present number of Kashmiri women are pursuing their career studies in various medical institutions of the state. Many Kashmiri women medics have till recently come back after achieving medical degrees from national and foreign medical institutions.

## REFERENCES

1. Khana S B. Evaluation of medical practice In Kashmir. Glimpses of Science and Kashmiri heritage 1st Ed. A.P.H. Publishing Corporation, New Delhi 2003: 61-74.
2. Kanpur M L. Educational position, In: Social and Economic History of Jammu and Kashmir, Anmol Publications, New Delhi: 201.
3. Kachwara J L. Problems of women's Education, In: Women's Education A conceptual Framework, A. P. H. Publishing Corporation, New Delhi: 168-170.
4. Dabla B. A. Changes Among the Kashmiri women, In: Working women in Kashmir, Rawat Publications, Jaipur: 1991: 25-26.
5. Shafi Aneesa. Working women in Kashmir, A. P. H. Publishing Corporation, New Delhi: 2002: 15.
6. Albuquerque U. Careers for women, In: Manorama year Book, Malayala Manorama Publishers, Malayalam: 2006: 472.
7. Madan T. N. The Image of the profession, In: Doctors and society, Vikas Publishing House, U. P: 1980: 275.
8. Narendran S. Women Achievers in the Medical Field, In: Beyond the Threshold Women on the move, B. R. Publishing Corporation, Delhi: 1995: 151.

Tel: 0194-2475723, Mob: 9419074233

## DEEN AND DEEN FINANCIAL SERVICES (P) Ltd.

Land Mark Shopping Mall, Karan Nagar, Srinagar, Kashmir

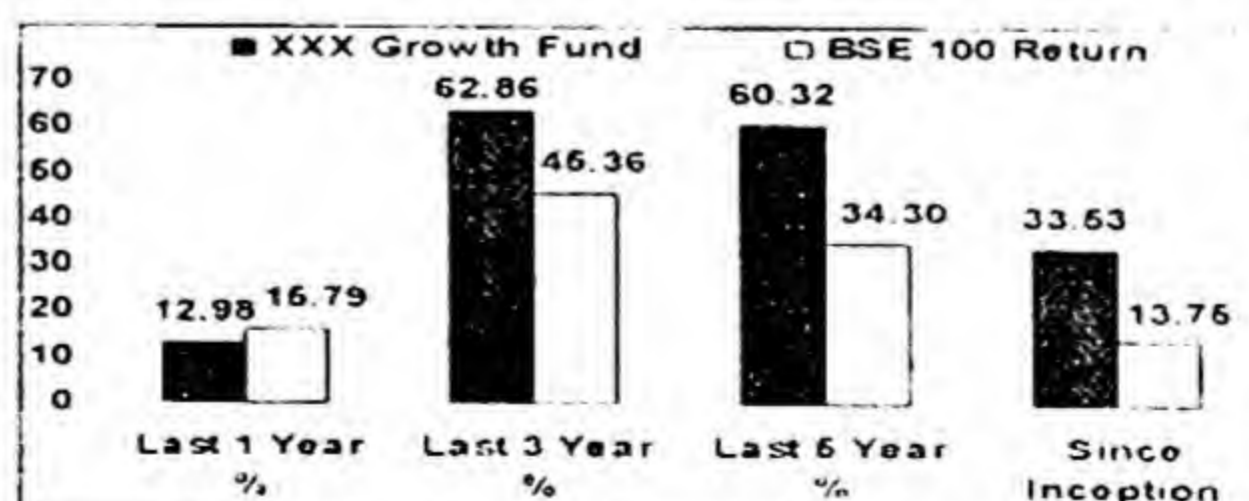
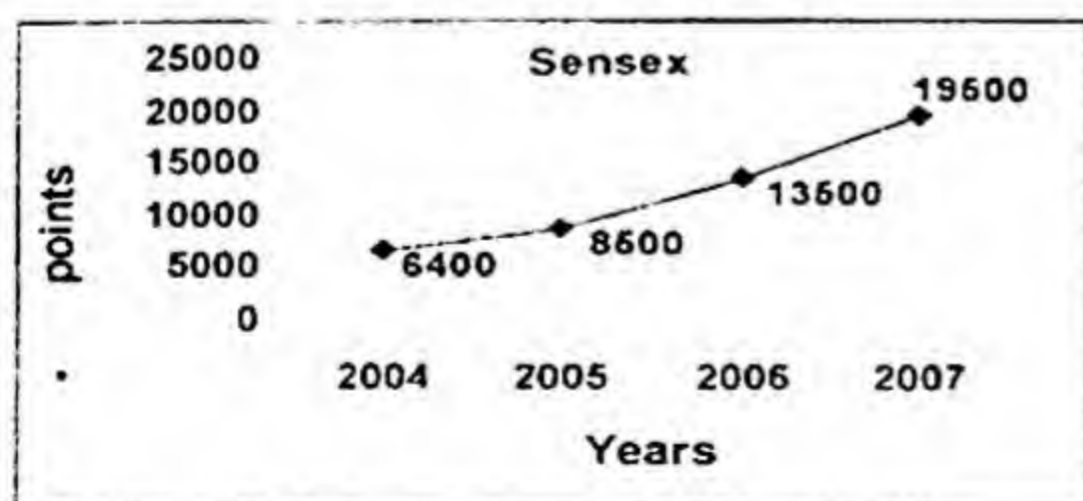
Near Dr. Allaqaband's Clinic

Share Stock Sub - Brokers\*

Registered With Securities & Exchange Board of India,  
National Stock Exchange & Bombay Stock Exchange

## Where You Can

1. Invest in shares of Blue Chip Companies engaged in sectors like Information Tech. (IT), Infrastructure, Media, Banks, Metals etc.
2. Invest in National & Multi National Mutual Funds of repute.



Investment in Shares and Mutual Funds is subject to market risks

\* Sub-brokers of LKP Shares and Securities P Ltd. Mumbai

# VITAL STATISTICS IN PULMONARY EMBOLISM

Tabassum Parvez HD.

## (PE : Pulmonary Embolism)

### Pulmonary Embolism

: Annual incidence of PE = 650,000 cases in USA annually.  
Incidence Rate : 1 in 418 or 0.24 % or 650,000 people in USA 54,166 / month , 12,500 / week, 1,780 / day , 74 / hour, 01 / per minute, 0 / second. Estimated 8.96 post operative P.E. occurred / 1000 surgical discharges.

### DEATH AND MORTALITY STATISTICS FOR PULMONARY EMBOLISM

750 / month , 173 / week , 24 / day , 1 / per hour

Deaths = 9000 / year

Death rate extrapolation for USA = 9000 / year

0 / minute , 0 / second.

### HOSPITALIZATION FOR PULMONARY EMBOLISM

66 % of Pulmonary Embolism required hospital admission in England in 2002 2003 ( Hospital episode Statistics, Department of health in England)(2002-2003 ) 44% of them were men and 36% were women. 91% PE required Emergency hospitalization., 11.5 days mean hospital stay for PE in England in 2002-2003. 64 was the mean age for Pulmonary Embolism Post operative Pulmonary embolism in hysterectomy cases studied for two year at Abbotabad Teaching Hospital and Ayub Medical College, was seen as 1.26% ( 4 out of 44 cases ).

1. Pulmonary Embolism is considered to be the 3rd most common cause of death.
2. Causes more deaths than Aids and Breast Cancer combined.
3. Experts believe it may contribute more than 100,000 per year.
4. Can effect young, fit and healthy people.
5. Often starts as a pain in the leg that does not go away.
6. Once clot is mobilized , death is imminent.
7. Caused by sitting cramped for a long time on bed, driving, flying.

### PHLEBOTHROMBOSIS AND PULMONARY EMBOLISM

Inspite of much research and publication remains dangerous and fatal complication of any operation

Single most lethal factor in post operative mortality of Gynaecological specialty.

One fatal pulmonary embolism occur in 1000 abdominal hysterectomy though unwelcome figure.

Vaginal hysterectomy reported to have greater immunity than abdominal hysterectomy .

Fatal Embolism seen to occur even after simple Dx. Curettage.

### CAUSATIVE FACTS :

1. Venous stasis : Static posture , confined to bed, lying on

their backs, bed clothes firmly tucked on lower limbs with minimum freedom of movement.

sitting immobile on a chair with popliteal veins at right angles to seat compresses leg veins in lowest dependant position. Remedy is walking with active movement of muscles.

2. In Anaesthetized patient dead weight of anaesthetized limbs act as compression and obstructing force. Operation itself genesis of post operative phlebothrombosis.
3. Alteration in blood coagulability : Increases in platelet count, decrease in clotting time, decrease fibrinolysis after operation. This defense mechanism is natural to counteract trauma of surgery. Increase blood coagulability as a response to injury, which acts by liberation of thromboplastin leading to DVT, Myocardial , Coronary and Cerebral thrombosis. Blood transfusion also increases blood coagulability, so a gap of few days ( a week ) should elapse between transfusion and operation

### .PATHOLOGICAL PROCESS

DVT in veins of soleus muscle where there is a series of vascular arcades and near the upper end of the thigh a confluence of large veins.

The thrombus gets dislodged and via venacava and right side of heart goes into the pulmonary arterial tree, depending on size and position of thrombus, different clinical pictures may be developed for example. Chest pain , Dyspnoea, Cyanosis, Plural rub, Pleural effusion,(Rt.) Ventricular strain, (Rt.) Bundle Branch Block. Thrombus rapidly grows , is friable, no local internal reaction because it is an aseptic process, no constitutional symptom or sign at first.Fatal pulmonary Embolism can occur even without a single symptom or sign and inspite of daily examination of lower limbs.

### DIAGNOSIS

Slight increase in temperature, pulse rate after 3rd Post Operative Day pain and itching in lower limbs, may on examination reveal a tender calf muscle, popliteal area or thigh. Dorsiflexion of ankle ( Homan's Sign ) elicits pain , very reliable screening by ultrasonic detector or thermograph. Radioisotope injections and phlebography to confirm Dx.

### PROPHYLACTIC TREATMENT.

1. Low mol weight dextrin infusion to decreases viscosity and sludding.
2. Low dose with Heparin subcutaneously.
3. Leg bandaging with early movements and ambulation.
4. Physiotherapy pre and post operatively. Regular exercise , regular breathing.

5. Anticoagulant Heparin 10,000 units / 4 hourly i.v. infusion:  
Once control is established, oral anticoagulant sodium Warfarin most popular agent . In case of over dosage bleeding, Antidote is protamine sulphate 4-5 ml counteract 10,000 units of Heparin. In case of dangerous hemorrhage . Fresh frozen plasma to restore clotting mechanism to normal, Other used are streptokinase (fibrinolytics).

Catheterization to the site of Embolus and injection of Normal saline at the site via it to break down or dissolve the clot.

### IN MASSIVE PULMONARY EMBOLISM

Pulmonary embolism using the "Smash and grab" Trendelburg's technique in who do not die but fail to respond to conservative treatment, though certainly lethal procedure .

### REFERENCES

1. Obstetrical pulmonary Embolism mortality United States , 1970-85.
2. Maternity mortality surveillance, United States , 1979-86.
3. Pregnancy related mortality due to Pulmonary Embolism in Guatemala, 1993-96.
4. Maternal mortality -1994. Trauma , pulmonary Embolism and maternal Cardiac diseases.
5. Deaths from Medical misadventure world diagnosis . Com. ( National Vital Statistics Report , CDC ,2003.
6. Patient safety in American Hospital, July, 2004. Health Grades.

with best compliments from:

# CAULSON & CO

1ST BRIDGE, SRINAGAR, KASHMIR TEL#: (0) 2472145  
(R) 2474355

E-MAIL: CAULSON@VSNL.COM



We Stock & Service  
Littmann Stethoscopes

Authorised Dealers of:

## **Johnson & Johnson Ltd.**

Ethicon, Codman, Endo, Hospital Products, Ortho & Ethnor Divisions

## **BIRLA 3M LTD.**

### **Stryker Products (USA)**

Manufacturers of

Laparoscopic, Arthoroscopic & Sinuascopic Equipments  
&

Equipments for all Types of Orthopedic, Neurological & Spinal Surgeries

# **The Changing Scenario in Rural Health : Sub-District Hospital Tangmarg, Distt. Baramulla opens a new page.**



Sub District hospital Tangmarg is a small beautiful hospital located in the north of Kashmir valley in the lap of world famous tourist resort, Gulmarg. It is a 30 bedded hospital with capacious lawns maintained by Gulmarg Development authority. Once upon the famous sanatorium of valley now caters to a large population and is guarded by tall slender pine and deodar trees.

The people in this part of valley are poor and have hard access to tertiary care hospitals in cities. In this regard Directorate of health services Kashmir came up with the programme of upgrading the infrastructure of this hospital and making necessary facilities available in this hospital to cater to the medical needs of the patients of this area who otherwise had to travel long distance to cities for the same.

Laparoscopic system of surgery was made available in this hospital in September 2006. The surgeon, paramedical staff and other personnel were trained to carry out the laparoscopic surgical procedures in this hospital. In this behalf a laparoscopic conference at SDH Tangmarg, first time in rural area was sponsored by directorate of health services Kashmir and organized by Dr. Mushtaq Chalkoo MBBS, MS, and FMAS. Consultant laparoscopic surgeon. The agenda of conference under title "LAPRO-2006" was a live operative workshop in which many laparoscopic surgeries were performed. Prof. Muneer Khan, HOD department of surgery, SMHS hospital performed Laparoscopic Cholecystectomy and laparoscopic appendicectomy.

Dr. Deep Goel, consultant laparoscopic colorectal surgeon Sir Ganga Ram hospital New Delhi performed laparoscopic mesh hernioplasty and demonstrated minimal invasive procedure for haemorrhoids, a latest technique for piles. Among other guests were the faculty members, postgraduates and registrars from SMHS, faculty from GMC Jammu and SKIMS.

Many surgeons from field participated in this conference. It was a mile stone in rural health. Since then laparoscopic procedures are routine in this hospital. The chief medical officer Baramulla inaugurated the function and distributed the mementos among the surgeons and the Participants

We have taken the dream of laparoscopic surgery to rural area first time in J&K State and in future advanced laparoscopic procedures will be possible in our small sub-district hospital Tangmarg.

**Dr. Mushtaq Chalkoo**  
MBBS, MS, FMAS  
Consultant Laparoscopic Surgeon  
Organiser "LAPRO-06"  
Email: - mushtaq\_chalkoo@rediffmail.com  
Cell No. 9419032292, 01942108415



**DR. MUSHTAQ CHALKOO-CONSULTANT LAPAROSCOPIC SURGEON  
SDH TANGMARG ORGANISING SECRETARY, "LAPRO-2006".**



**PROFESSOR MUNEER KHAN, HOD SURGERY SMHS HOSPITAL,  
DR N.A. SALROO & DR. NIGHAT CHAURING THE SESSION FOR OPERATIVE  
WORKSHOP**



**DR. DEEP GOEL, CONSULTANT COLORECTAL  
LAPAROSCOPIC SURGEON  
SIR GANGA RAM HOSPITAL NEW DELHI PERFORMING  
LAPAROSCOPIC MESH HERNIOPLASTY**



**PARTICIPANTS AND DELEGATES OF (LAPRO-2006)**